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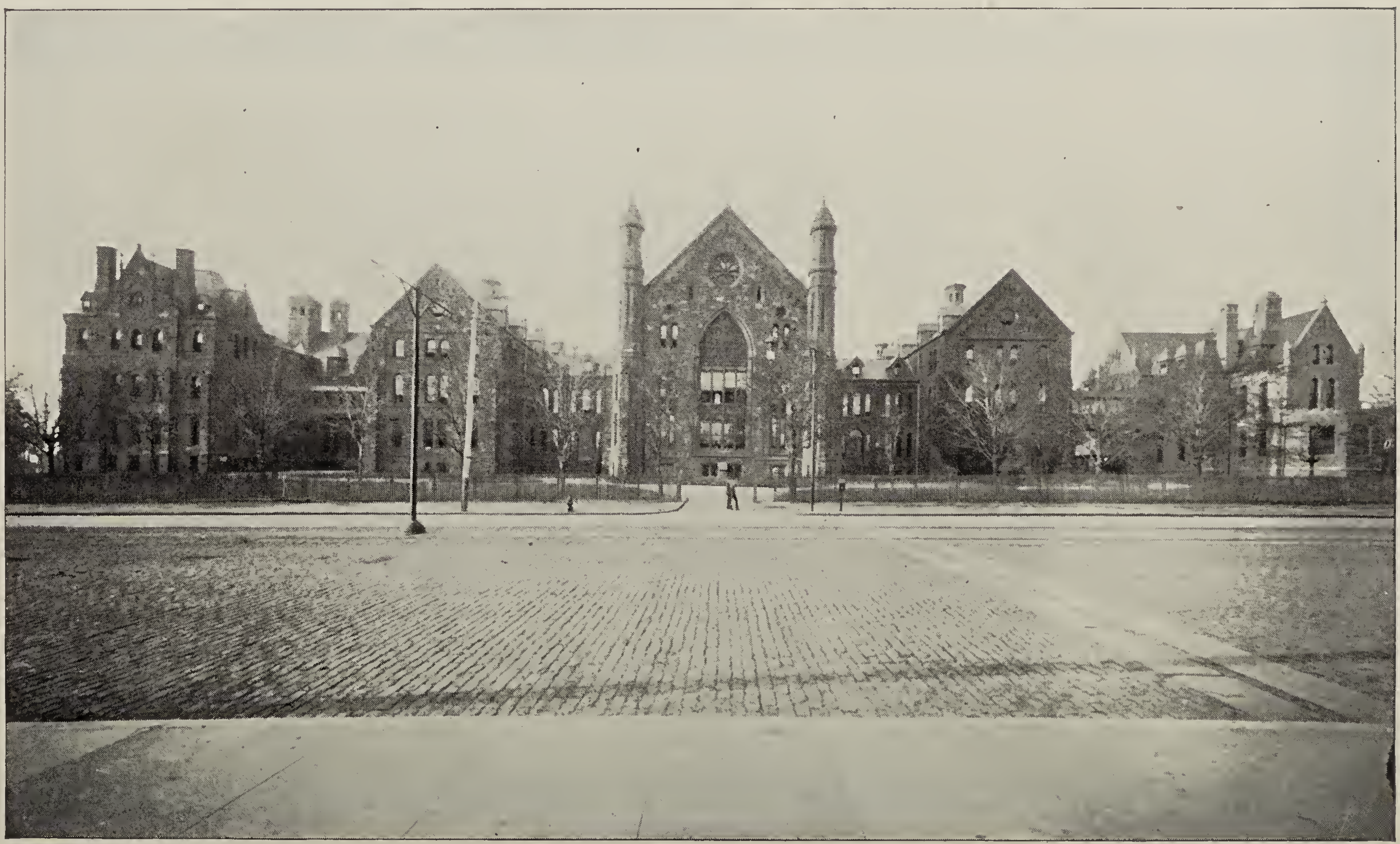

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HOSPITAL

OF

THE PROTESTANT EPISCOPAL CHURCH
IN PHILADELPHIA

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# MEDICAL AND SURGICAL REPORTS

OF THE

# EPISCOPAL HOSPITAL

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*VOLUME III*

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PHILADELPHIA
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1915

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Astley P. C. Ashhurst, M.D., *Chairman.*

This volume has been edited by
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John Crerar Library, Publications. Chicago, Illinois.

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Johns Hopkins Hospital Bulletin. Baltimore, Maryland.

Massachusetts General Hospital, Collected Papers. Boston, Massachusetts.

Münchener medicinische Wochenschrift. Munich, Germany.

Philippine Journal of Science, Medical Section. Manila, P. I.

Pilcher Hospital, Bulletin of the. Brooklyn, New York.

Presbyterian Hospital Reports. New York, N. Y.

Roosevelt Hospital, Medical and Surgical Reports. New York, N. Y.

Royal College of Surgeons of England, Calendar. London, England.

University of Missouri Bulletin, Medical Series. Columbia, Mo.

Westminster Hospital Reports. London, England.

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# PAPERS BASED ON WORK DONE IN THE EPISCOPAL HOSPITAL, 1914-1915

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## From the Medical Department of the Hospital

*From Dr. Stevens's Service.*

Stevens: The Hyperchlorhydric Syndrome; Pathogenesis and Treatment.

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Longaker: The Use of Silk Ligaments and Tenodesis in Infantile Paralysis.

### FROM THE DERMATOLOGICAL DEPARTMENT OF THE HOSPITAL

Walker: Keratosis Palmaris and Plantaris.

### FROM THE DEPARTMENT OF DENTAL SURGERY

Cameron: Report of the Department for 1914.

# FROM THE REPORTS OF THE SUPERINTENDENT FOR 1914 AND 1915

| | 1914 | 1915[1] |
|---|---|---|
| Patients admitted | 5922 | 4534 |
| Patients remaining from previous year | 390 | 283 |
| Total number treated during year | 6312 | 4817 |
| Patients discharged | 5545 | 4089 |
| Patients died | 484 | 408 |
| Patients remaining December 31 | 283 | 320 |
| Total | 6312 | 4817 |
| Average daily number of patients in the hospital | 415⅔ | 309½ |
| Capacity of Hospital (Beds) | 500 | 500 |
| Ambulance calls | 1435 | 1306 |
| Ambulance accident calls | 371 | 422 |
| Total | 1806 | 1728 |
| Average daily number of ambulance calls | 4.95 | 4.75 |
| Average duration of treatment of in-patients, excluding those in Harrison Memorial House for Incurables | 19¾ | 19 days |
| Cost of patient per day[2] | $1.5175 | $1.67 |
| Gross cost of patient per day (calculated on total expenditures of all kinds) | 1.9266 | 1.94 |
| New Patients treated in the Dispensary: | | |
| Medical | 6,930 | 4,310 |
| Surgical | 8,226 | 6,641 |
| Eye | 3,638 | 2,769 |
| Ear, Nose and Throat | 3,219 | 2,344 |
| Skin | 1,056 | 758 |
| Orthopedic | 518 | 469 |
| Genito-urinary | 308 | 255 |
| Dental | 1,049 | 1,067 |
| Receiving Ward | 7,362 | 7,219 |
| Total | 32,306 | 25,832 |
| Average daily number of Dispensary Patients | 345 | 260 |
| Average daily number of Emergency Cases treated in Receiving Ward | 20.17 | 19.80 |

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[1] The reduction (during 1915) in numbers of admissions to the various departments is largely due to the institution on January 1, 1915, of a system of charges to all patients able to pay.

[2] Cost of patient per day, as distinguished from gross cost, means the deduction of $12,000 for the Dispensaries and the deduction also of the Improvement and Repair Account. These figures include all patients admitted to the wards.

# HISTORY OF ANESTHESIA[1]

By ELLIS E. W. GIVEN, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

THE desire for insensibility to pain during surgical procedures dates almost to the earliest operation. Different drugs were made use of and various methods adopted by the early nations in their attempts to render the subject of any of the painful surgical operations free of all or even a part of the suffering incident to clumsy cuttings and manipulations.

The discovery of the methods for the production of anesthesia, both partial and complete, and the associated muscular relaxation accompanying some of these states, was only a link in the chain of surgical progress, having its origin in the dim and distant past; and only by the progressive advancement of all the sciences was it possible for this chain, with its links of pharmacy, physiology, anatomy, and chemistry, to be developed; yet with all of their individual development it remained for dentistry and medicine to take these separate links and so weld them that this chain of surgical progress, the pride of the world in this our twentieth century, could securely hold from suffering and death many thousands of our brethren who are daily called to depend upon the workers in and with this chain.

Interesting indeed is the study of the methods employed many hundreds of years ago for the amelioration or relief of pain. At the siege of Troy the surgeons used carbonic acid gas and alcohol for the relief of the wounded, and Nestor used a medicated poultice "composed of cheese, onions, and meal, mixed with the wine of Pramos," when treating the wounded Machaon.[1] Patroclus,[2] when treating the wounded thigh of Euryphylus:

---

[1] Read before the Episcopal Hospital Clinical Society, January 18, 1915.

> "Cut out the biting shaft; and from the wound
> With tepid water cleansed the clotted blood;
> Then, pounded in his hands, the root applied
> Astringent, anodyne, which all his pain
> Allay'd; the wound was dried, and stanched the blood."

The Assyrians are credited with being the first to practice digital compression of the carotid arteries to bring about anesthesia before doing the operation of circumcision.[3] [4] It is probably from the practising of this method of producing anesthesia the artery received its name "carotid," and interesting in this connection is the fact that the literal translation of the Greek and Russian terms for carotid is "the artery of sleep."

The Egyptians used cannabis indica, which they called "hashish," and the juice of the poppy to bring on a drowsy condition before attempting painful procedures; but as cannabis indica was indigenous to China, the Chinese were in all probability the first to use it to produce a dulling of the sensations. It is recorded[5] that the Chinese physician Hoa-Tho, who lived about 225 A.D., gave a preparation of hemp to a patient whereby he became "as if he had been drunk or deprived of life," but in a few days recovered fully "without having experienced the slightest pain during the operation."

The Romans brought from Memphis a species of powdered rock,[1] [2] which when moistened with sour wine and applied to wound, would produce local anesthesia.

---

[1] Artificial Anesthesia and Anesthetics, Lyman, 1881.

[2] Iliad.

[3] Leçons sur les Anesthesiques et sur l'Asphyxie, Claude Bernard, 1875.

[4] C. Hoffmann: De Thorace, lib. 2, cap. xxix, Francof., 1627 in fol., p. 77.

[5] Comptes Rendus de l'Acad. de Sc., 1849, tom. xxviii, p. 195.

Herodotus,[3] who lived about 484 B.C., makes mention of the Scythians using a vapor of hempseed to cause drunkenness by inhalation. The vapor was produced by throwing the herb, which was probably cannabis indica, into the fire or on to stones heated for the purpose.

Theodoric referred,[4] in 1490, to a preparation used by his father, Hugo of Locca, which he termed "oleum de lateribus, a most powerful caustic and a soporific, which, by means of smelling alone could put patients to sleep on occasion of painful operations which they were to suffer." On another occasion the following appears: "In operations for hernia, for instance, he teaches that the patient must be tied to the bench or table with three bands, one around the ankles, another around the thighs, and a third across the chest holding the arms and hands," which indicates that this "oleum" was not made use of on all occasions where cutting was required. He later describes the making of this preparation, which is elsewhere called "spongia somnifera," as follows: "Take of opium, of the juice of the unripe mulberry, of hyoscyamus, of the juice of hemlock, of the juice of the leaves of mandragora, of the juice of the woody ivy, of the juice of the forest mulberry, of the seeds of lettuce, of the seeds of dock, which has large round apples, and of the water hemlock each an ounce. Mix all of these in a brazen vessel, and then place in it a new sponge; let the whole boil as long as the sun lasts on the dog days, until the sponge consumes it all, and has boiled away in it. . . . As oft as there shall be need of it, place this sponge in hot water for an hour, and let it be applied to the nostrils of him who is to be operated on until he has fallen asleep, and so let the surgery be performed. This being finished, in order to awake him, apply another sponge, dipped in vinegar, frequently to his nose, or throw the juice of the root of fenugreek[1] into the nostrils; shortly he awakes."

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[1] Pliny.

[2] Probably powdered marble, which when mixed with acid in the form of the "sour wine" produced carbon dioxide gas.

[3] Carey's Herodotus, book I, 202.

[4] Lib. iv, cap. 8 (quoted by Snow).

In 1555 John-Baptista Porta described the making of volatile substances "which were converted into an essence which had to be kept in leaden vessels, hermetically sealed, to prevent the escape of its volatile parts. . . . When required for use the cork was removed and the flask was applied to the nostrils of the patient, who inhaled the volatile essence till his senses became, as it were, locked up in a citadel, and buried in a sleep so profound that only by the greatest violence could he be aroused. After waking from such a sleep there was no uneasiness in the head, nor any recollection of what had taken place."[2]

Efforts toward the alleviation of pain were being made in Italy as early as 1589, and it is recorded[3] that Giambattista Porta, who practised in Naples, used a combination of hyoscyamus, solanum, poppy, and belladonna prepared in a closed vessel and at the time of operation, "the lid being opened the patient would draw in by breathing the most subtle strength of the vapor, so that thereby he would be buried in a most profound sleep nor be aware of what had been done to him."

Du Bartas,[4] a poet of the middle of the sixteenth century, records in verse the practice of that time to produce some degree of insensibility to pain during surgical procedures in the following verse:

> "Even as a surgeon minding off to cut
> Some cureless limb, before in use he put
> His violent engines on the vicious member,
> Bringeth his patient in a senseless slumber,
> And griefless then (guided by use and art)
> To save the whole, cuts off the infected part."

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[1] Trigonella Foenum-Graecum, which is an European annual leguminous plant cultivated at present in France and Germany for its seeds.

[2] Quoted by Lyman.

[3] Sir Frederick W. Hewitt, M.V.D., Anesthetics, 4th ed.

[4] Sylvester translation, 1592.

The teachings of Dioscorides that the sleep produced by mandragora wine would last four hours or more is probably responsible for the statement of Friar Laurence, whom Shakespeare causes to say:

> "Through all thy veins shall run
> A cold and drowsy humor, which shall seize
> Each vital spirit; for no pulse shall keep
> His natural progress, but surcease to beat:
> No warmth, no breath, shall testify thou livest;
> The roses in thy lips and cheeks shall fade
> To paley ashes: thy eyes' windows fall
> Like death when he shuts up the day of life:
> Each part deprived of supple government,
> Shall stiff and stark, and cold, appear like death
> Thou shalt remain full two and forty hours,
> And then awake as from a pleasant sleep."

And again in "Cymbeline,"[1] which takes date of about 1613, Shakespeare causes Cornelius, the court physician, when responding to the request of the Queen for some poisonous drug, to mention both animal experimentation and artificial sleep in his remarks to the audience, in the following words:

> "Will stupify and dull the sense awhile,
> Which first perchance she'll prove on cats and dogs
> Then afterwards up higher; but there is
> No danger in what show of death it makes,
> More than the locking up of spirits for a time,
> To be more fresh reviving."

With these crude attempts to produce insensibility to pain, it was necessary for the early workers in surgery and the patients as well to be content; but the links in the chain of surgical progress were gradually being formed, and as a result there was selected from the different plants, mandragora, which was the most potent drug known to the ancients. Cuneiform characters and Egyptian hieroglyphics referring to mandragora, which was known as "Phallus of the Field," appear as early as 3000 B.C., and testify to the use of this plant for medicinal purposes as an anodyne and anesthetic.

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[1] Cymbeline, Act I, scene vi.

The first botanical work[1] mentioning mandragora directs attention to its property of inducing sleep and of its use as an aphrodisiac. Thus "Aphrodite to assuage her grief for the death of Adonis is said to have thrown herself on a bed of lettuce and mandragora."[2]

Apuleius,[3] writing of the use of the drug in the surgery of that day, states that one-half ounce of the wine taken by mouth was sufficient to render the person insensible to the pain of amputation. Aetius, a Greek physician, writing about the fifth century and referring to an overdose of mandragora, said "danger was present when the patient kept constantly drawing in air through his mouth, gasping for breath; and if help was not soon afforded he died convulsed."[4]

Dioscorides[5] [6] writing of mandrogora in A.D. 100, refers to this pain-allaying drug, and records that "some persons boil the root in wine down to a third part and administer a cyathus[7] of the decoction to those in want of sleep, or (*suffering with*) severe pains of any sort, and also before operations with the knife or actual cautery, that they may not be felt." He also gives the following directions for making the wine: "A wine is prepared from the bark of the root without boiling, and three pounds of it are put into a cadus[8] of sweet wine, and three cyathi of this are given to those who are required to be cut or cauterized, when being thrown into a deep sleep they do not feel any pain."

Pliny,[9] who lived 23 to 79 A.D., states the juice from the leaves is more powerful than the preparation made from the root, and that "before cuttings and puncturings . . .

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[1] Theophrastus.

[2] Anesthetics, Antient and Modern, Burroughs, Wellcome & Co.

[3] Lib. vii, p. 207.

[4] Surgical Anesthesia, Bellamy Gardner.

[5] De Med. Mat., bk. iv, 76.

[6] Brit. and For. Medico-Chir. Rev., 1874, vol. liii, p. 242.

[7] Somewhat more than one and one-half ounces.

[8] About eighteen gallons.

[9] Lib. xxv, cap. 13.

it is sufficient for some persons to have sought sleep from the smell." Particular mention is made of the drug's usefulness in allaying pains, Apuleius[1] writing "if anyone is to have a limb mutilated, burnt, or sawn, he may drink half an ounce with wine, and whilst he sleeps the member may be cut off without pain or sense." In fact, mandragora is so frequently referred to by the early writers that it undoubtedly was held in high repute for the allaying of suffering, and very probably it was wine made from this drug and then mingled with myrrh which the kind-hearted women offered to our Saviour while on the cross, but which he did not accept, as it was the common practice in the early part of the first century to offer to those condemned to death on the cross this mandragora wine.[2]

Such continued faith in mandragora and so many references to its usefulness in the early practice of surgery lead to a desire to know more about this pain-allaying drug. The Greeks called the plant "Circeum,"[3] believing it to be possessed of an evil spirit and that when it was gathered it gave forth such terrible shrieks that no one hearing it could live. On this account special directions were issued for the collection of the root. One ancient writer thus directs: "To gather ye mandragora, go forthe at dead of nyght and take a dogge or other animal and tye hym wyth a corde unto ye plante. Leese ye earth round about ye roots, then leave hym, for in hys struggles to free hymself he will teare up ye roote, whych by its dreadfull cryes wyll kyll ye animal."[4]

In the United States the drug is unknown as a remedy, but referred to as Mandragora Officinarum L. (Mandragore, Fr.; Alraunwurzel, G.) It is a perennial European plant, having a spindle-shaped root, often forked below, which caused it to be frequently compared in form to that of the human figure. In early times the belief was held that there were actually male and female species. The root was accredited with aphrodisiac properties, and portions of it were worn by the Babylonians to prevent sterility as early as 2000 B.C.

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[1] De Herbarum Virtutibus, cap. 131.

[2] Bull. of the Medico-Legal Society, N. Y., 1881, vol. iv, Nos. 7 and 8.

[3] Derived from "Circe," the witch.

[4] Anesthetics, Antient and Modern, Burroughs, Wellcome & Co., London.

The plant is poisonous and narcotic, belonging to the same family as belladonna. An alkaloid, mandrogorine, was isolated by Crouzel.[1] "Mandragorine is colorless, inodorous, deliquescent, melts at from 77° to 79° C., and has the formula $C_{17}H_{23}NO_3$. It seems to be isomeric with atropine, but is not converted into it by alkaloids. The sulphate and the hydrochloride are crystalline and deliquescent. A second alkaloid in much smaller amount was also extracted, of which the gold and platinum double chlorides were formed. Both alkaloids had a mydriatic action."[2]

"R. Schaurer[3] states that in addition to the already known alkaloids, of which the root . . . contains 0.376 per cent., another toxic substance is present; this appears to be allied to a fat; it is extremely poisonous to frogs in very minute doses. It is insoluble in water; readily dissolved by petroleum ether and other fat solvents. Heat and saponification do not appear to lessen its toxicity. When administered in very minute doses to frogs it occasions very profuse hemorrhagic diathesis of the pharyngeal mucous membrane. Hemorrhage also occurs under the skin at the site of injection, and pigmentation in the neighborhood is destroyed. In animals with warm blood these characteristic general reactions do not follow hypodermic administration of the poison. In them much inflammation and edema of the injected region is produced, followed by necrosis of the tissues."[4]

In 1874 Dr. Richardson[5] reported some experiments he had made as to the efficacy of mandragora. Obtaining a specimen of the root five centuries old, he powdered it, and using a weak spirit allowed the root to remain in contact with the spirit for several weeks. A very strong tincture was thus obtained.

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[1] Pharmaceutical Journal, vol. xv, 3d S., p. 1067.

[2] F. B. Ahrens, Berlin, 1889, p. 2159–2161.

[3] Pharmaceutical Journal, No. 2667, p. 731.

[4] Merck's Report, January, 1915.

[5] Brit. and For. Medico-Chir. Review, 1874, vol. liii, p. 242.

Tests carried out with this tincture by mouth and by subcutaneous injection proved it narcotic. In birds, deep sleep resembling that of chloral was produced. Five minims of the above tincture of the root, subcutaneously administered to an animal, or ten minims by mouth, produced sleep "broken by short and peculiar excitement and wildness," which passed into a disturbed sleep and then a gradual recovery. With large doses the deep sleep ended in death due to failure of the respiration. The heart was scarcely influenced by the early respiratory failure, but continued to beat ("in one instance for seven minutes") after the respiration ceased. Postmortem findings showed congestion of the lungs with an excess of fluid in the bronchial tubes.

"On the human subject the agent has a very potent action. The tincture benumbs the tongue when a little is placed upon it, and the taste of the substance, with the peculiar sensation that is induced by it, lasts for many hours . . . A dose of twenty minims, insufficient to produce comatose symptoms, was exceedingly potent in effect. It caused a desire for sleep, a sense of fulness in the vessels of the brain, a peculiar confused vision, an exaggeration of sounds, and a curious excitability, akin to hysteria . . . these symptoms were not removed for a day, and they left some lingering uneasiness and coldness longer . . . As after atropine, there was dilation of the pupil from mandragora."

The doses used by Dr. Richardson were very much smaller than those used by the ancients when they desired to produce insensibility to the pains of operations or of the cautery, and it may be that the smallness of his doses was the reason he did not get more profound symptoms from the drug. He stated that his experiments bore out in all particulars the claims of the early workers with the drug.

In 1800 Sir Humphry Davy[1] suggested that the inhalation of a gas would produce insensibility to pain during surgical operations in the following words:

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[1] Researches, Chemical and Physical, 1800.

"As nitrous oxide gas in its extensive operations appears capable of destroying physical pain, it may probably be used with advantage during surgical operations in which no great effusion of blood takes place."

And again in 1818 the suggestion, yea more, the fact of surgical etherization, was stated[1] "when the vapor of ether mixed with common air is inhaled it produces effects very similar to those occasioned by nitrous oxide. . . . It is necessary to use caution in making experiments of this kind. By the imprudent inspiration of ether a gentleman was thrown into a very lethargic state, which continued with occasional periods of intermission for more than thirty hours, and was accompanied with great depression of spirits; for many days the pulse was so much lowered that considerable fear was entertained for his life."

No practical use was made of these suggestions, and they were apparently lost sight of by the profession, for patients were subjected repeatedly to long and severe operations with no other preparation than the administration of large doses of laudanum followed by alcohol in one or another form up to complete intoxication, or by bleeding the patient to the point of fainting, this latter method being used especially in the reduction of dislocations. For the reduction of dislocation of the hip, reliance was commonly placed on the depressing effect caused by the injection into the rectum of a strong infusion of tobacco. This usually produced within a few minutes depression and relaxation to a degree favorable for reduction, but the reduction of the dislocation being accomplished, sometimes within a few minutes, the depressing effect of the tobacco continued for an hour or more with at times alarming symptoms.[2]

Three methods were used[3] to overcome muscular resistance when operations were undertaken on the eyeball, throat, or rectum: (*a*) free bleeding, (*b*) use of tartrate of antimony, (*c*) tobacco by rectal injection, these were all carried to the point of depression and relaxation of the vital forces; but while consciousness remained there were no methods known to any of the operators that could completely abolish these muscular efforts. One instance is recorded[1] of a lady who, desiring to have teeth extracted, received five hundred drops of laudanum in forty-five minutes.

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[1] Anonymous statement, but is accredited to Faraday.

[2] Boston Med. and Surg. Jour., 1897, vol. cxxxvi, No. 1, p. 2.

[3] Ibid.

Dr. Benjamin Eddie Cotting, writing[2] under the head of "A Bit of Professional Reminiscence," so pathetically describes an amputation of the breast in the pre-ether days that the following is quoted at length:

"Soon after the discovery of ether I performed a similar operation for a patient who declined to take ether; she knew she could bear it without flinching, and did not wish to have her mind clouded or her senses benumbed. The first cut—a rather long one—was borne pretty well; but the second was too much for her. She raved and stormed, roared out heavy groans and heartrending shrieks. The operation would have had to be abandoned had not sufficient muscular force been provided for quietly in advance. She was held during tetanic-like spasms, and the operation was completed while she was in a position almost that of opisthotonos. Her outcries aroused the neighbors, who rushed in in numbers to know what the occasion might be. In due time she recovered, but she never ceased to regret her mistake. Her case was often used in advancing anesthesia, and did much to establish the administration of ether in the neighborhood."

Such was the condition of affairs in the first half of the nineteenth century; the profession, and general public as well, desiring but hardly daring to hope for the day when necessary painful procedures could be undertaken without the dread with which both were wont to look upon all such procedures.

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[1] Trials of a Public Benefactor, Nathan P. Rice, M.D.

[2] Boston Med. and Surg. Jour., January, 1897, vol. cxxxvi, p. 1.

A study of the history of the discovery of the anesthetic properties of ether and its introduction to the profession as an anesthetic brings into prominence the names of many individuals in both the medical and dental professions, but two of these shine out clearly and distinctly as those to whom belongs the full credit for this important and epoch-making discovery. I refer to Dr. Crawford W. Long and Dr. W. T. G. Morton. These two gentlemen having confidence in the drug, accepted both the moral and legal responsibility imposed by their experiments and brought the administration of ether by inhalation to so high a state that the present advanced methods of modern medicine have little to add (when using ether alone) to the technic used by the discoverers over fifty years ago, and both of them deserve lasting credit and praise for their efforts.

Dr. Crawford W. Long was the person who first employed ether as an anesthetic for surgical operations, and the circumstances which led him to believe that ether administered by inhalation would prevent pain during surgical operations are of much interest.

Dr. Crawford W. Long was born in Danielsville, Georgia, November 1, 1815[1]; at the age of nineteen he graduated second in his class from Franklin College. From the medical department of the University of Pennsylvania he was graduated in 1839, after which he entered one of the New York hospitals as resident physician, where he made a good name for himself as a surgeon. After finishing his course in this hospital he returned home at the request of his father, to practice medicine in a country district.

The practice of inhaling nitrous oxide gas at evening companies for its exhilarating effects was very common among the young people living in the neighborhood of Dr. Long's home, and on one occasion he was requested to prepare some of the gas for this purpose. His response was he did not have the necessary apparatus to prepare the gas, but "has an article (sulphuric ether) which would produce equally exhilaratory effects and was as safe." This substance was accepted, and proving satisfactory for such purposes, became quite popular; and he further remarks in his original statement:[1] "On numerous occasions I inhaled ether for its exhilaratory properties, and would frequently, at some short time subsequent to its inhalation, discover bruised or painful spots on my person which I had no recollection of causing, and which I felt satisfied were caused while under the influence of ether. I noticed my friends while etherized received falls and blows which I believed were sufficient to produce pain in a person not in a state of anesthesia, and on questioning them they assured me that they did not feel the least pain from these accidents. Observing these facts I was led to believe that anesthesia was produced by the inhalation of ether, and that its use would be applicable in surgical operations."

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[1] Johns Hopkins Hosp. Bull., No. 77–78 (1897).

Dr. Long's first opportunity to make practical application in surgery of his belief in ether anesthesia was on March 30, 1842, when he removed a small tumor from the neck of Mr. James M. Venables after telling him that the operation would probably be painless if he would first breathe the ether which he administered on a towel, and the patient continued to breathe the ether during the operation. Thus we see that Dr. Long as early as the spring of 1842 had used ether for the relief of pain during surgical operations and had made a report of such use to the Georgia State Medical Society; but as the Society at that early date published no transactions its proceedings were not made public and the discovery of ether anesthesia was not announced to the great body of the medical profession; but had Dr. Long sent a copy of his report to any of the more important medical journals his discovery would have been made known to, and tried by many of the profession, and undoubtedly he would have been hailed by the entire civilized world as the first discoverer of the anesthetic properties of ether, for, as will be shown later, his belief in, use of, and public announcement to a regularly organized medical society preceded by exactly four and one-half years that of Dr. W. T. G. Morton, who on September 30, 1846, extracted a tooth under its influence; his first public demonstration at the Massachusetts General Hospital was a few days later, being October 15, 1846.

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[1] Paper read before the Georgia State Medical Society, 1842.

Dr. Long refrained from publishing his discovery, not from any sordid or selfish reason, but only as he said[1] he might "by etherization in a sufficient number of cases fully satisfy my mind." This waiting to satisfy his mind of the worth of his discovery, and to permit in the meantime hundreds of persons to suffer untold agonies under the operating knife, was undoubtedly the cause of his not receiving universal recognition as the only discoverer of this, one of the most important discoveries of all ages, ranking equally high with Lister's far-reaching discovery of the principles of antisepsis, for how could these principles of antisepsis be successfully applied to a struggling, howling patient?

Dr. W. T. G. Morton, to whom the world is lastingly indebted for the practical introduction of ether as an anesthetic, was born in Charlton, Worcester County Massachusetts, August 19, 1819.[2] He was very studious as a youth, but was compelled to divide his time between his education and the regular routine work of the ordinary New England farm on which he spent his early years.

In 1840 he entered the Baltimore College of Dental Surgery, and in two years, following his graduation in dentistry, he opened an office in partnership with Dr. Wells, a brother dentist, but the partnership not resulting in entire financial success, they dissolved it by mutual agreement in the fall of 1843,[1] and Dr. Morton opened his own office in Boston,[2] and later he matriculated at the Medical School of Harvard University. He was not graduated in medicine from this institution, as the demands made upon his time following the discovery of the anesthetic properties of ether by inhalation required that he discontinue his studies.

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[1] Paper read before the Georgia State Medical Society, 1842.

[2] The Bostonian, William E. Hayden, M.D., vol. iii, No. 4, p. 315.

Dr. Morton was the recipient of the honorary degree of Doctor of Medicine from Washington University in 1849.

Dr. Morton, who appears to have been of an inventive mind, discovered a method for the insertion of artificial teeth, but as this method necessitated the removal of the root of the diseased one, and this being a very painful procedure, he was especially desirous of inventing a method for the painless extraction of teeth, and experimented with this in mind; but not until September 30, 1846, did he make use of ether by inhalation. On this date he told a patient who came to his office to have a very painful tooth extracted, and who being afraid of the pain incident thereto had asked to be mesmerized, he had something better, and saturating a handkerchief with ether, he gave it to the patient to inhale the vapor. Sufficient insensibility soon developed for the extraction of a firmly rooted bicuspid tooth. The patient recovered consciousness in a few minutes, and said he had no knowledge of what had taken place. Newspaper reporters probably were as energetic at that time as they are at the present, for the following appeared in the *Boston Daily Journal* of October 1, 1846:

"Last evening, as we are informed by a gentleman who witnessed the operation, an ulcerated tooth was extracted from the mouth of an individual without giving the slightest pain. He was put into a kind of sleep, by inhaling a preparation, the effects of which lasted for about three-quarters of a minute, just long enough to extract the tooth."

This notice appearing in the daily paper seems commonplace enough at this late day, but appearing at that time it was one of the most important that had ever occurred, for it gave notice in few words that another forward stride had been taken in the world's progress, and that henceforth all who require painful operations may have the same performed while sleeping under the trained eye of a trained anesthetist.

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[1] Trials of a Public Benefactor, Nathan P. Rice, M.D.

[2] History of Medicine, Francis Randolph Packard, M.D.

Many acrimonious discussions are recorded as having taken place about this time as to the amount of credit due Dr. Morton; the Senate of the United States spent many hours trying to unravel the problem as to the amount of credit due the doctor, but it is sufficient for the present purpose to record that Dr. Morton made his first public demonstration of the inhalation of ether for the alleviation of pain during surgical operations at the Massachusetts General Hospital on October 15, 1846, at which time Dr. Warren removed a tumor from the neck of a male patient. That the profession was decidedly incredible at the time Dr. Morton made this demonstration may be inferred from the remarks of Dr. Warren when he announced to those present "a test of some preparation was to be made for which the astonishing claim had been made that it would render the person operated on free from pain."[1]

On the day appointed for this first public demonstration Dr. Morton was a little late in arriving at the operating room on account of his having been delayed in arranging the apparatus with which he was going to administer the ether and Dr. Warren remarked with sarcasm, "As Dr. Morton has not arrived, I presume he is otherwise engaged," seeming to intend to convey the impression that the doctor was not going to be present to perform his promised demonstration. But almost at the time these sarcastic words were being spoken, Dr. Morton walked into the operating room, whereupon he was greeted with the words, "Your patient is ready," and was met with the cold, unsympathetic looks of all of the medical audience. The patient selected for this demonstration was a man who had presented himself for the removal of a tumor from the side of his neck. At once turning to the patient, who was an entire stranger to him, Dr. Morton spent a little time in soothing him, dispelling his fears of impending pain or danger, thus gaining his confidence, coöperation, and good-will; teaching a very important lesson indeed—one which it would be well were all our present-day anesthetists to heed. The ether was poured on a towel and the vapor inhaled from that as it was held over the nose and mouth. When sufficient of the drug had been inhaled, Dr. Morton announced to Dr. Warren, "Your patient is ready," and it is easy of belief that emphasis was placed upon the *first* word of the sentence, considering the sarcasm and hostility with which he was greeted upon his entrance a few minutes earlier. After the operation the patient declared he had felt no pain, and Dr. Warren turned to those present and said, "Gentlemen, this is no humbug!"

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[1] Trials of a Public Benefactor.

From this time onward the administration of ether by inhalation rapidly became the accepted method of preparing patients for surgical operations.

Dr. Morton was granted a patent November 12, 1846, for his pain-destroying drug, which he had named "Letheon," and he was soon compelled to discontinue his studies in the Medical School of Harvard University, as the demand was so great for his services as an anesthetist. It was not possible to keep the identity of this "Letheon" a secret, as wherever used the odor proclaimed its true name, and it was not long before the entire country cast aside all restraint caused by the patent right, and unhesitating use was made of ether wherever painful operations were required. The medical officers of the United States Army and Navy, themselves members of that Government which had granted a "patent," disregarded the immunity conferred and made unhesitating use of the drug.

The name "anesthesia" and also "anesthetic" were suggested to Dr. Morton by Dr. Oliver Wendell Holmes in a personal letter, a part of which is quoted:

Boston, November 21, 1846.

"My dear Sir:

Everybody wants to have a hand in a great discovery. All I will do is to give you a hint or two as to names, or a name, to be applied to the state produced or the agent. The state should I think be called "anæsthesia." This signifies insensibility, more particularly to objects of touch. The adjective will be "anæsthetic." Thus we might say the state of anæsthesia, or the anæsthetic state. The means would be properly called the antiæsthetic agent. Perhaps it might be allowable to say anæsthetic agent, but this admits of question."

It will be recalled that Dr. Long four and one-half years earlier had used the term "anesthesia" and "anesthetic," thus having within his grasp this great discovery together with its accepted name had he but published the facts to the world in time.

It is interesting to note that the two gentlemen to whom thus belongs the credit of the discovery of the anesthetic property of ether and its successful introduction into surgical practice, made first use of the drug exactly four and one-half years apart (March 30, 1842—September 30, 1846); that in both instances, the patients were males, and that each patient submitted to the operation for the removal of a tumor from the neck; that one of the discoverers represented the Southern, the other the Northern section of the United States (the two great sections of our country at that time); that one was a member of the organized (city) dental profession, the other a humble country doctor; one a graduate in medicine[1] the other in dentistry,[2] and both of them in the active practice of their respective professions, one in a large city the other as a quiet country doctor; thus showing that neither the medical nor the dental professions, nor any one section of our great country, nor any one institution can claim the entire credit for this far-reaching discovery and its successful introduction to the surgery of the world.

[1] University of Pennsylvania, 1839.

[2] Baltimore College of Dental Surgery, 1840.

# VARIOUS TYPES OF LUES

## A CLINICAL AND LABORATORY STUDY. THE ABELIN REACTION. DIRECT SUBDURAL INJECTIONS[1]

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THE clinical history of syphilis, admittedly one of the most protean of all diseases, is in the process of being rewritten, and when the chapters are relatively complete it will be found that we are indebted rather to the laboratory worker than either the internist or neurologist for our more complete knowledge. It was at one time supposed that syphilis of the nervous system was a late manifestation of the disease, and this statement is generally true from the clinical point of view. Precocious syphilis was the term employed by Gowers for those cases in which patients manifest organic nervous phenomena within a year from the appearance of the primary lesion. For many years it has been recognized as a possibility, and cases have been reported by Gowers, Fournier, Oppenheim, Nonné and others, including an instance furnished by Gregory and Karpas of a case of cerebral syphilis occurrring six months after the development of the primary lesion.

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[1] Read before the Section on Practice of Medicine at the Sixty-fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914. Reprinted from the Journal of the American Medical Association, January 16, 1915, vol. lxiv, pp. 199–202.

In going over some of the patients and records in the hospital at the naval home in Philadelphia, with Dr. Stokes, formerly Surgeon-General of the U. S. Navy, I was struck with the greater virulence of the disease in general and the serious and occasionally early involvement of the nervous system occurring in sailors who had contracted the disease in the Orient. It is solely from the clinical point of view, however, that we of today are justified in regarding syphilis of the nervous system as among the later phases of the activity of the treponema. Soon after the use of salvarsan became general, considerable discussion arose as to the part it apparently played in causing involvement of various cranial nerves and other more or less pronounced nervous symptoms. Previously free, involvement became manifest within a few hours or days following an injection of the drug. Routine study of the cerebrospinal fluid from luetic patients revealed to Dreyfus that about 80 per cent. of late primary and early secondary cases show distinctive pathologic changes in the spinal fluid (cytologic increase or increase in protein content and more rarely a positive Wassermann). Thus, the explanation of the preceding, essentially a Herxheimer reaction, local or diffuse, depends on distribution of the treponemas, and is due to liberation of toxins resulting from the lytic action of salvarsan on the parasite.

This carries with it the thought, first, that all initial doses should be small, and secondly, if symptoms of involvement of the nervous system occur, it is not necessarily a contraindication to the further use of the remedy, but rather the reverse, though in small doses. Even the few early cases which we have felt justified in submitting as illustrative, show well the pathologic changes in the spinal fluid and there is absolutely no doubt from the point of view of the serologist, that the vast majority of luetics, from the date of the primary infection, are essentially candidates for syphilis of the nervous system. Though they may not present a single symptom, the treponemas invade all parts of the body within a day or two at most from the time of infection. Neisser succceeded in inoculating animals with blood taken from monkeys five days after the original infection and with splenic pulp taken on the twelfth day. Hoffmann has shown that monkeys may be infected by using the blood from a human in the secondary stage, and that under such circumstances the incubation stage in the monkey is shortened. Infection successively passaged through a series of animals increases the virulence of the infection and shortens the incubation period. Flügel recovered the treponema from the blood of a woman with a secondary, maculopapular eruption. Indeed, it is probable that the eruption is in the nature of a parasitic embolism similar to the rose spots of typhoid fever, which are mycotic, congested, capillary loops. Veillon and Girard selected a typical maculopapule and excised it on the third day after it appeared. The capillaries were dilated and congested with an early, perivascular, lymphocytic infiltration, and treponemas were found, by the silver method, in and about the capillaries. Neisser, Buschke, Levaditi, Tomasczewski, Noguchi and others have found the parasite in tertiary lesions of the skin and various organs including the nervous tissue, either by direct staining methods or by inoculation into animals. Von Zeissl found records of more than 150 cases of facial paralysis, deafness or retinitis occurring within one year of the period of original infection. Thus it is evident that every stage of the disease is potentially infective; the possibility of transmission, however, becoming less in direct proportion to the elapsed time since the original infection.

Two points in connection with the primary infection, the knowledge that syphilis may be contracted without the development of a chancre, and the well-known evanescence of secondaries in some cases, should render us tolerant in our judgment of those from whom no history is obtainable. Jonathan Hutchinson has reported several cases, one in a nurse and several in physicians, in whom the disease followed a punctured wound, the first evidences of actual infection being adenopathy and eruption. This possibility has since been confirmed experimentally. A chancre is only possible when cutaneous or mucous membrane infection occurs. Again, we sometimes see a patient in whom a chancre has developed, often atypical, to be sure, who admits exposure months or even a longer period in the past, but who denies recent infection. Keogh, in 1911, called attention to the fact that, in relapses occurring even at long periods after salvarsan has been given, whether the Wassermann be positive or negative at the time of the relapse, the clinical picture is more or less a replica of the condition presented at the time the patient first sought treatment. Thus, if treatment is given in the primary stage, a relapse is often manifested as a chancre, secondary stage—secondaries. Keogh regards this as a true latency, unlike the apparent latency of cases treated with mercury, which, as a rule assume a far more advanced type in the relapse.

Briefly, then, the present concept of syphilis is that, in its earlier stages, it is a septicemia with more or less involvement of the nervous structures and coverings from the beginning. In the course of time, for some unknown reason, special predilection is manifested for some particular organ or tissue, after a period of apparent latency which only serologic study will reveal. In some instances, indeed, the latency is so well concealed that only a provocative Wassermann, as first suggested by Gennerich and almost simultaneously by Milian will reveal it. This test is practically a Herxheimer reaction and proves the presence of treponemas somewhere in the body hence their lysis and the subsequent successful application of serologic diagnosis. Recognizing the comparative frequency of early involvement of the central nervous system, as shown by the serologist, it would seem advisable to make studies not alone of the serum, but also, in every case of lues or suspected lues, to make routine examination of the spinal fluid, and to control treatment by subsequent studies, and, finally, to determine a cure by provocative injections governed by complete laboratory observations. It is not too much to claim that such a plan would in all probability prevent the development of the more destructive forms at present so common among us.

As to types of lues, it is only possible to speak in very general terms. Except for the rare cases in which a chancre fails to develop and those in which secondaries are fleeting or suppressed, the most constant and fairly uniform stage is the septicemic or secondary period. Beyond this we may speak of pernicious anemic or other blood dyscrasias, of cardiovascular, visceral, osseous, ulcerative cutaneous, central nervous (including special sense-organs) and congenital types, but each of these groups presents many varieties. Serologically, we are also unable to speak of definite types. We believe, however, that when a standard technique is developed, especially with reference to the Wassermann reaction, and particularly when we reach a conclusion as to the proper antigen to use, less criticism will be leveled against its diagnostic value than is unfortunately the case at present. Even under existing conditions, the chief critics are those who possess no laboratory experience. Until such time it might be of value, in reporting laboratory results, to mention the antigen employed and the particular form of complement-deviation test used in the work, and especially the former. Using an alcoholic antigen prepared from fetal syphilitic liver, a sheep hemolytic system and the method as given by Citron, only varying the complementary strength to obtain relative quantitative results instead of using the continental method of a definite 0.1 c.c. of guinea-pig's serum, it will be found that the large majority of late primary cases, and all untreated secondary cases will give a positive serum, and very rarely a secondary will give a positive Wassermann in the spinal fluid. The majority of secondary cases will also show some other spinal fluid change, as increase in protein content or an increase in cell-count, with prompt reduction of Fehling's solution. Latent cases occasionally give negative Wassermann in the serum and fluid, with a positive Wassermann in the serum as the rule, though they may give changes in the protein content of the fluid; but in these cases a reaction may be invariably provoked. The majority of non-latent tertiary cases give positive serums and more marked pleocytosis or protein increase and occasionally a positive spinal fluid Wassermann. Of the nervous forms, tabes is the most frequent. A very small number is serologically negative, but these are rare. The majority have a positive serum, a positive Wassermann in spinal fluid, a moderate pleocytosis and a more or less marked Nonné and less often a positive Noguchi. Since we have used spinal fluid in amounts of 0.5, 1.0, and 1.5 c.c., we have rarely had negative Wassermanns. In the cases clinically marked by severe pain, the cell-count is usually higher, and other evidence of inflammation of the root-zones and meninges is shown by marked Nonné or Ross-Jones and Noguchi reactions. A large proportion of this type also gives a positive Wassermann in the spinal fluid. When strong positive Wassermann is obtained in both serum and spinal fluid, in most instances there is a more or less pronounced pleocytosis up to 50 or 60 possibly per cubic millimeter, with protein increase and often a positive Lange (gold solution). These are the least amenable to treatment and are spoken of as Wassermann-fast types, though not by any means invariably so.

The unilateral type of tabes and those cases presenting but one or two symptoms, as pupillary rigidity and Westphal's symptom, present no special serologic type differing from any of the above. There is one clinical phenomenon of great value diagnostically, which occurs in the vast majority of tabetics and one which is not sought for routinely, but which furnishes strong collateral proof of the existence of tabes often at a time when very few criteria are manifest. We refer to that which Collier Martin has termed the tabetic sphincter. Digital examination of the rectum reveals external sphincter paresis. Firm lateral pressure is painless and results in more or less relaxation of the anus, permitting a good view of the lower part of the rectum. This relaxation may not be overcome for a period from a few seconds to some minutes.

The greater the degree of meningeal involvement the higher the cell-count and the greater the protein content. Hence, in cerebrospinal syphilis, the high count is found except in the purely endarteritic forms. In some of the cerebrospinal cases the serologic picture closely simulates that of paresis, except that it is far more amenable to treatment than is paresis. Hence a serologic diagnosis is of value not alone in a diagnostic sense, but also from the prognostic standpoint.

In general paresis the rule is to find a strongly positive Wassermann in both serum and spinal fluid, a moderate pleocytosis and a marked protein reaction. Until very late in the course of the disease, the serum is always positive, but in a small proportion of cases the spinal fluid Wassermann may be negative throughout. Paresis frequently falls under the Wassermann-fast group, and in most instances the Lange (gold) solution runs a parallel course with the Wassermann of the spinal fluid. These laboratory results and our failure to make satisfactory serologic or clinical impress on the types of lues involving the nervous system, when combined therapy by mouth and intravenously was employed, led us to take up subdural work. We had made attempts to recover phenolsuphonephthalein, methylene blue and formaldehyde from the spinal fluid but without success, after previous administration subcutaneously in the first, by mouth and subcutaneously in the second, and by mouth (hexamethylenamin) in the third instance, and we had never observed an icteric spinal fluid in any case of jaundice. We were, therefore, ready to accept the view that the best if not the only way to reach the nervous tissue is by direct methods. We found, too, that when we used a direct subdural injection of neosalvarsan without a previous intravenous injection, we were able to recover arsenic in the urine in a few hours, showing that absorption had certainly taken place. Tilney, before the New York Neurological Association, reported some experimental evidence along these lines. He used trypan-blue, a vital stain, injecting 0.1 per cent. into cats and allowing them to live for twenty-four hours. At the expiration of that time, when a subdural injection had been given, the dura mater was intensely stained, the brain stained diffusely. The viscera were not stained. Microscopically he found the trabeculæ of the pia extending into the brain and cord were stained, also the walls of the bloodvessels. The ganglion-cells and nerve-fibers were not stained. It seemed, therefore, that the dye found its way into the nervous tissue along the trabeculæ and by the bloodvessels. Intra-arterial injection caused skin and visceral staining but none in the brain or cord or the meninges. Intravenous injection resulted in staining of the dura, pia and pituitary, though less than by intraspinal injections. Frozen sections showed the cord unstained. Subcutaneous injection resulted similarly to the arterial, the nervous tissues were unstained, but the viscera were stained.

Sicard employed ink by the lumbar subdural route in animals. In from two to four hours he found particles in the cerebellar region and still later in the region of the peduncles and within the ventricles. In from twelve to fourteen hours he found them also in the cortex of the cerebrum. He observed also that substances in solution in the cerebral fluid were removed by osmosis while particulate substances were removed by leukocytic phagocytosis. In spinal anesthesia it is well known that, if the dose be large and the specific gravity of the injected fluid be low, within a brief period, often a matter of a few minutes, both respiratory disturbance and vomiting will occur. We know also that the subdural fluid and that of the ventricles communicate normally through the foramen of Magendie at the apex of the fourth ventricle and through the foramina of Luschka at the outer margins of this same ventricle. Both on anatomic grounds, therefore, and on the basis of experimental evidence we are justified in our belief that better results must ensue in early cases of syphilis of the nervous system if subdural treatment is resorted to; and as many of the cases also have more or less cutaneous, visceral or osseous lues, the combined intravenous and subdural routes seem to be indicated in those whose infection during the earlier period has not been studied and treated in sufficient detail to prevent the more serious manifestations from arising.

To arrive at the approximate dosage of neosalvarsan given subdurally when 12 c.c. of a 40 per cent. solution are employed after an intravenous injection of 0.9 gm., we took the figures of Bischoff, who found the total volume of blood in the bodies of two executed criminals to be 7.1 per cent. and 7.7 per cent. respectively. Thus, to a patient weighing 150 pounds, each cubic centimeter of serum would contain about 0.00017 mg., and 12 c.c. about 0.002 mg. We then employed the Abelin reaction to determine the presence of arsenic in serum and urine. Using dilutions of known strength we obtained the following figures.

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|---|---|
| 1:100 | Beautiful red contact ring |
| 1:10000 | Reddish ring |
| 1:20000 | Brownish ring |
| 1:30000 | Brownish ring |
| 1:40000 | Brownish ring |
| 1:50000 | Very faint brownish ring |
| 1:60000 | Negative |

We were unable to obtain a reaction in the serum of blood removed in ninety minutes, in any patient treated intravenously, and we have found a negative in one case and weakly positive in another at the end of sixty minutes. In the urine we were unable to recover it after ten hours. After intramuscular injections we did not examine the serum, but in the urine we obtained the reaction in thirty-one and forty-eight hours respectively. Abelin found practically the same results, though in two patients of his series treated intravenously, he obtained the reaction in the urine in seventeen and twenty-two hours. We found the reaction positive in the serum in from ten to fifteen minutes after the intravenous injection was given. The early disappearance of the Abelin reaction led us to take the blood in from thirty to forty-five minutes after the intravenous injection was completed. We have treated nine cases in all, two by the Swift and Ellis alone, two by the direct method alone, and five by combined Swift and Ellis and direct methods. Despite the experience of Wechselmann and Marinesco and more recently the experimental evidence of Swift and Ellis, we determined to try the effect of direct injections without previous intravenous. The maximum dose employed was 0.02 gm. of neosalvarsan, and the minimum 0.006 gm.

In all dosage we found the direct method too irritating. After a total of 11 direct injections, we found that the three paretics suffered the least, two of them having to be catheterized, one during the first twenty-four hours, one only at the end of the third day, and the paretic who had received 0.02 gm. and 0.01 gm. at different times, had no symptoms at all. Of the cases of taboparesis and tabes, all the direct injections were followed by severe pain, causing retention necessitating the use of the catheter for as much as two weeks and obstinate constipation. The direct injections were given in salt solution to one patient only, while in the others we employed the patients' inactivated serum in salt solution, a 40 per cent. dilution. It is well known that irritative phenomena occur after the Swift and Ellis method, so that if, as Ravaut claims, hypertonic salt solution robs the direct injection of the irritating effects of neosalvarsan, it might be well to use hypertonic salt solution as a basis for the dilution in the method of Swift and Ellis. One very advanced case of taboparesis, No. 28 of our series, was treated by the method of Swift and Ellis and by the direct method at various times. The patient finally died. Grossly, a little congestion of the meninges was found at necropsy, but sections studied by W. G. Spiller failed to show definite evidence of irritation.

One case of paresis treated by both the direct and indirect methods, No. 34, shows marked improvement after failure to make any impress by seven intravenous injections given at weekly intervals and a number of injections of sodium cacodylate in 15 grain doses. The other two paretics are *in statu quo*. Two of the tabetics have shown marked improvement clinically, Nos. 26 and 27. They have received but two Swift and Ellis injections each. On the other hand, we have had two cases of tabes not included in our series, one with absent knee-jerks and lightning pains, the other with pupillary rigidity, absent knee-jerks, vomiting without pain, paresthesia and tabetic sphincter, both of whom recovered under intravenous treatment and mercury and iodids by mouth. The former, less marked, made an absolute recovery clinically and serologically, the latter has become subjectively symptom-free, knee-jerks now being present by reinforcement, vomiting, paresthesia and lightning pains totally relieved, the pupillary rigidity uninfluenced, serum now negative, but spinal fluid has not as yet been examined. These cases prove our previous statement that if recognized early, and this includes careful clinical and serologic study, tabes may prove amenable to intravenous injections together with mercury and iodids by mouth. In later cases, the value of the Swift and Ellis and direct methods are still *sub judice*.

## Report of Specific Cases

### SECONDARY, TERTIARY AND CONGENITAL WITHOUT CEREBROSPINAL SYMPTOMS

Case 1.—P. F., aged 18. Chancre on lip of about four weeks' duration. Was treated at once with mercury by mouth. Has a diffuse maculopapular rash. Wassermann of serum 4 plus; luetin, negative.

Spinal fluid: Cell-count and Nonné, negative; Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.5, 1.0 and 1.5 c.c., all dilutions negative.

Abelin Reaction: Blood-serum after 0.5 gm. neosalvarsan intravenously, positive after fifteen and thirty minutes was weakly positive after sixty minutes and after ninety minutes negative.

Washed blood-clot. Was laked and an attempt made, by means of the Abelin reaction, to determine the presence or absence of arsenic. Unsuccessful owing to the color similarity of laked blood and Abelin reaction.

Case 2.—M. S., aged 30. Chancre on penis five weeks ago. Has been taking pills. Has an indurated scar on foreskin. General adenopathy. Maculopapular eruption on chest. Wassermann of serum, 4 plus.

Spinal fluid: Cell count, 10 lymphocytes; Nonné and Noguchi, negative; slight reduction of Fehling's; Wassermann, 0.2 and 0.4 c.c., negative in all dilutions.

Phenosulphonephthalein Test: First hour, 15 per cent.; second hour, 37 per cent.

Abelin Reaction: Intravenous neosalvarsan given November 13, 1913. Blood-serum after 0.5 gm. neosalvarsan intravenously, positive after fifteen and thirty minutes, was weakly positive after sixty minutes and negative after ninety minutes.

Urine: Markedly positive in three and one-half hours; weakly positive in five and one-half hours; negative in eight hours. After 0.3 gm. neosalvarsan intramuscularly, given November 23, 1913. was weakly positive in one hour, positive in three, weakly positive in five, positive in eight, twelve, and twenty-four hours, weakly positive in thirty-one, negative in thirty-three hours.

Case 3.—M. N., aged 20. History of chancre five months ago, followed by eruption and constitutional symptoms. Has been taking pills ever since. Has mucous patches. General adenopathy. Scar on foreskin. Wassermann of serum, 4 plus; luetin, negative.

Spinal fluid: Cell-count, 20 lymphocytes; Nonné and Noguchi, negative; Fehling's reduced; Wassermann, 0.5, 1.0, and 1.5 c.c., negative in all dilutions.

Case 4.—L. M., aged 24. History of suppurating sores on penis one year ago, followed by slight rash and constitutional symptoms at which time he took pills for two months. Now has a diffuse inflammation of throat. Wassermann of serum, 4 plus; luetin, negative.

Spinal fluid: Cell-count, negative; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.5, 1.0, and 1.5 c.c., negative in all dilutions.

Case 5.—T. B., aged 19. Chancre of five weeks' duration. Has a maculopapular rash on chest and abdomen. General adenopathy. Wassermann of serum, 4 plus.

Spinal fluid: Cell-count, negative; Nonné and Noguchi, negative; Fehling's reduced; Wasserman, 0.2, 0.4 and 0.8 c.c., negative in all dilutions.

Case 6.—J. S., aged 48. Denies knowledge of chancre on penis. Has been taking pills for rash on body last ten days. Has syphilitic iritis. General adenopathy. Profuse maculopapular eruption, large papular and papulonodular syphiloderms. Examination revealed typical chancre on penis. Wassermann of serum, 4 plus; luetin, positive (pustule).

Spinal fluid: Cell-count, negative; Nonné and Noguchi, negative; Fehling's reduced; Wassermann, 0.5, 1.0 and 1.5 c.c., negative in all dilutions.

Case 7.—T. B. aged 38. A patient with well-defined chronic diffuse nephritis. Denies venereal history. No previous treatment. Has a papular rash on arm and leg. Wassermann of serum, 4 plus.

Phenolsulphonephthalein Test: Percentage first hour, 0; second hour, 5, on admission. Convulsion same day. After purges and sweats, thyroid gland and Fischer's enteroclysis, first hour, 15 per cent.; second hour, 10 per cent.

Spinal fluid: Cell-count, 30 lymphocytes; Nonné and Noguchi, negative; Fehling's reduced; Wassermann anticomplementary reactions. Six months later, during which time mercury by mouth was given, Wassermann of serum had gradually become negative; luetin, negative.

Spinal fluid: Cell-count, negative; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.5 c.c.—, 1.0 c.c.—, 1.5 c.c.—.

Phenolsulphonephthalein Test: After a few months' treatment, first hour, 25 per cent., second hour, 27 per cent.

Case 8.—T. P., aged 30. Chancre on penis, 1903. Mercurial treatment the following eight months. Neosalvarsan at another hospital, March 1914, and later in the Episcopal Hospital. For past year has been complaining of hoarseness and cough. Has a tertiary syphilitic involvement of nose and throat. Had been taking mercurous iodid. Wassermann of serum, negative. Wassermann made six weeks after second intravenous of neosalvarsan, during which time protiodid was given. Luetin, positive (papule).

Spinal fluid: Cell-count: 20 lymphocytes; Nonné and Noguchi, slightly positive Fehling's reduced; Wasserman, 0.5 1.0 and 1.5 c.c., negative in all dilutions.

Case 9.—O. P., aged 42. Gumma of liver. Family history, negative. No history of previous illness. Denies veneral infection.

*Present Illness.*—Was well prior to seven weeks ago, at which time he developed diarrhea, vomiting, indigestion and ascites. Lost 13 pounds in weight.

*Physical Examination.*—Liver enlarged from fourth interspace to 5 cm. above the umbilicus, is nodular. Left lobe larger, more nodular and extended farther down than right lobe. Ascites, 4.4 liters of fluid withdrawn which later recurred and 4.2 liters were removed. Wassermann of serum, 4 plus. Phthalein test, 30 per cent. the first hour; second hour, 20 per cent. Gastric analysis showed a hyperacidity. Urine contained a trace of albumin and no casts. Mercury administered by inunctions. Potassium iodid was given; 0.45 gm. of neosalvarsan injected intravenously and another injection of 0.6 gm. intramuscularly was given some days later. Patient markedly improved, the liver reduced in size. Fluid did not reaccumulate. Gained weight, felt subjectively better.

Case 10.—J. H., aged 43. For some years subject to sore throat. Underweight. Always cold. Multiple lesions on penis nineteen years ago. Promptly recovered; no internal treatment. August, 1913, developed dull pain in epigastric region without reference to meal. Sense of discomfort in bending and unable to sleep on left side on account of sense of weight in epigastrium. Gastric analysis, normal.

*Physical Examination.*—Irregular enlargement of left lobe of liver, extending down almost to transverse umbilical line. Spleen not enlarged. Diagnosis, gumma. Moderate general adenopathy. Absent knee-jerks even on reinforcement. No urinary phenomena, no lightning pains. No paresthesias, no Romberg. No ataxia.

Dec. 2, 1913: Wassermann of serum, 4 plus.

Dec. 7, 1913: Intravenous 0.6 gm. salvarsan. Protiodid then given.

Dec. 18, 1913: 0.3 neosalvarsan intramuscularly. (Gumma much reduced in size.)

Dec. 29, 1913: Patient has gained considerable weight and has lost all muscular pains promptly, considered by him rheumatic. 0.3 gm. neosalvarsan intramuscularly. Protiodid has been continued all this time.

Jan. 17, 1914: Gumma totally disappeared. Liver normal in outline. No longer feels cold; 0.9 gm. neosalvarsan intravenously. Beginning Jan. 24, 1914, a series of hypodermatic injections of sodium cacodylate in 10-grain doses, eight in all, given. Feb. 28, 1914, Wassermann of serum, 4 plus. Inunctions then started and continued intermittently for some weeks, with salivation on several occasions. All medicine then stopped for three weeks and May 13, 1914, Wassermann of serum, 3¾ plus.

Spinal fluid: Cell-count negative; Nonné and Noguchi slightly positive; Fehling's reduced; Wassermann, 0.5, 1.0 and 1.5 c.c., all negative.

CASE 11.—F. R., tobacco salesman, aged 66. Family history, negative. Suppurative sore on penis fifteen years ago. Treated with mercury for three years.

*Present Illness.*—For past six months has complained of weakness, shortness of breath, dizziness. Recently lost 30 pounds in weight. No indigestion. No loss of blood.

*Physical Examination.*—Patient is poorly nourished. Skin is of a lemon color. Muscles soft and flabby. No bleeding areas. No apparent focus of infection. Old scar on penis, evidence of an old destructive lesion. Urine: Specific gravity, 1010, a cloud of albumin, hyaline and granular casts. Gastric analysis: Free hydrochloric acid, 0; total acidity, 40; lactic acid, negative. Stools: ova and occult blood negative. Blood picture: Red blood-cells, 740,000; white blood-cells, 6000; hemoglobin, 35 per cent. Differential count: Polymorphonuclears, 70 per cent.; mononuclears, 8 per cent.; transitional, 2 per cent.; lymphocytes, 17 per cent.; eosinophils, 3 per cent. Poikilocytosis and marked anisocytosis. No nucleated reds. Wassermann blood-serum, 4 plus. Given 0.2 gm. of neosalvarsan intravenously, iron administered. One month later, blood-picture was: Red blood-cells, 1,480,000; white blood-cells, 4880; hemoglobin, 40 per cent. Differential count: Polymorphonuclears, 48.6 per cent.; mononuclears, 1.6 per cent.; lymphocytes, 49.3 per cent.; eosinophils, 0.66 per cent. No nucleated reds. Poikilocytosis and marked anisocytosis. Mixed treatment administered. Nine months later patient was markedly improved. Appears well. Feels subjectively well. Gained weight. Free from symptoms. Normal blood-picture after six months and no further evidence of achylia gastrica. Wassermann of serum made at that time, 3 plus. Considering himself well, he refused further treatment, but has recently developed an iritis.

CASE 12.—M. McD., aged 20. A case of congenital syphilis. Has paralysis of left leg from an attack of anterior poliomyelitis when a child. Necrosis of the left toes. Wassermann of serum, 4 plus; luetin, positive (papule and vesicle).

Spinal fluid: Cell-count, Nonné and Noguchi, negative. Fehling's reduced. Wassermann, 0.5, 1.0 and 1.5 c.c., negative in all dilutions.

Case 13.—H. K., aged 30. Congenital syphilis. Interstitial keratitis. Has always been in good health. Has typical physiognomy of hereditary syphilis. Hutchinson's teeth. Severe interstitial keratitis in the left eye. Wassermann of serum, 3 plus; luetin, positive (pustule).

Spinal fluid: Cell-count, negative; Nonné and Noguchi, positive; Fehling's reduced; Wassermann, 0.2 and 0.4 c.c., negative in all dilutions.

Case 14.—B. G., aged 14. Admitted to the Episcopal Hospital under the provisional diagnosis of splenomedullary leukemia. Family history, parents living and well. One brother stillborn, one sister died at six months of marasmus. Patient was always delicate and undernourished. Had mumps and pertussis in childhood, typhoid fever at 11. Frequent attacks of sore throat. Complaints on admission, headache pain in both costal regions, dizziness, constipation, enlargement of abdomen.

*Physical Examination.*—Pupils, normal. Heart and lungs negative. Liver extends from fourth interspace, midclavicular line, almost to transverse umbilical line. Surface uneven. Spleen extends 1 inch below transverse umbilical line and to within ½-inch of a vertical line drawn through umbilicus.

Blood-count: Red blood-cells, 4,600,000; white blood-cells, 7120; hemoglobin, 44 per cent. Differential: Polymorphonuclears, 66 per cent.; mononuclears, 1 per cent.; transitional, 3 per cent.; lymphocytes, 24 per cent.; eosinophiles, 2 per cent. and 4 per cent. of an atypical leukocyte which we were unable to identify. This led to a Wassermann of the blood-serum, which was positive. The patient's father's and mother's blood-serum were then taken and both were found positive, the latter less marked than that of the father or child. The child had a moderate adenopathy, including the epitrochlear glands. Wassermann of serum still 4 plus after three intravenous, each 0.3 gm., salvarsan. Mixed treatment for six months following; Wassermann of serum then 2 plus. Gain in weight, 38 pounds. Blood-picture normal. Liver normal size. Spleen half the original size. Subjectively well.

## TABETIC CASE HISTORIES[1]

Case 15.—C. T., aged 38. Denies infection. Denies previous treatment. Initial symptoms one year ago were attacks of gastric pain and vomiting. Complains now of ataxia, imperfect control of bladder and bowels, shooting pains in legs, girdle sensation. Is emaciated, has unequal Argyll Robertson pupils. Fundus: gray optic nerves, concentric contraction of all the fields of vision. Ataxic gait. Romberg's sign, positive. Ataxia of left arm. Impaired sensation of left leg. Loss of patellar reflexes. Wassermann of serum, negative; luetin, negative.

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[1] None of the cases in this group was treated subdurally.

Spinal fluid: Cell-count, 50 lymphocytes; Nonné and Noguchi, positive; Fehling's slightly reduced; Wassermann, 0.5, 1.0 and 1.5 c.c., 4 plus, in all.

Case 16.—A. B., aged 55. Tabes. Chancre when 22 years old. Took mercury by mouth for six months thereafter. First complained of ataxia four years prior to being seen. Has recently complained of shooting pains in the legs, anesthesia and paresthesia. Imperfect control of bowels. Pupils are dilated, immobile both to light and accommodation. Romberg's sign positive. Impairment of sensation. Ataxia of limbs. Relaxed sphincter ani. Loss of patellar reflexes. Wassermann of serum, 4 plus; luetin, negative.

Spinal fluid: Cell-count, negative; Nonné and Noguchi, negative; Fehling's reduced; Wassermann, 0.2, 0.4 and 0.8 c.c., negative in all dilutions.

Case 17.—W. A., aged 28. Tabes. Chancre in 1901. Is uncertain as to treatment. Complains only of attacks of gastric pain and vomiting for past year. Is emaciated. Pupils irregular, reacting sluggishly to light. No fundus changes. Romberg's sign suggestive. Sluggish patellar reflexes. Good tone to sphincter ani. Wassermann of serum, 4 plus; luetin, positive (papule and vesicle).

Spinal fluid: Cell-count, 50 lymphocytes; Nonné and Noguchi, very slightly positive; Fehling's reduced; Wassermann, 0.5 c.c.—$3\frac{1}{2}$ plus, 1.0 c.c. and 1.5 c.c.—4 plus.

Case 18.—M. E., aged 45. Tabes. Charcot's joint. Denies infection and knowledge of any previous treatment. First complaint was of pain and swelling of right ankle four years ago. Paresthesia of lower limbs. Loss of sexual desire, at times diplopia. Is emaciated. Pupils negative. Ataxic gait. Romberg's sign positive. Impairment of sensation in lower limbs. Patellar reflexes absent. Well-defined Charcot's joint right ankle. Wassermann of serum, 4 plus.

Spinal fluid: Cell-count, 60 lymphocytes; Nonné and Noguchi, positive; Fehling's not reduced, contained blood; Wassermann, 0.2 c.c. plus, 0.4 c.c. 3 plus, 0.8 c.c. 4 plus.

CASE 19.—C. M., aged 52. Tabes. Chancre in 1906. Treated with mercury for a short time. Mild secondaries. Complains of weakness in legs and tendency to fall. Is uncertain as to time of onset. Pains in legs, anesthesia and paresthesia. Imperfect control of bladder. Pupils react sluggishly to light. Ataxic gait. Romberg's sign positive. Patellar reflexes absent. Wassermann of serum negative.

Spinal fluid: Cell-count, 250 cells; Nonné positive; Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.5 c.c. negative, 1.0 c.c. $\frac{1}{2}$ plus, 1.5 c.c. 2 plus.

CASE 20.—W. B., aged 39. Tabes. Denies infection. Only complaint is shooting pains in legs for past month. Has Argyll-Robertson pupils. Beginning optic atrophy. Ataxic gait. Romberg's sign positive. Loss of patellar reflexes. Wassermann of serum, negative; luetin, positive (papule).

Spinal fluid: Cell-count, 30 lymphocytes; Nonné and Noguchi, positive; Fehling's, insufficient spinal fluid; Wassermann, 0.5, 1.0 and 1.5 c.c., 4 plus in all.

CASE 21.—R. K., aged 57. Tabes. Chancre forty-two years ago. Treated with mercury for about six weeks. Complains of poor vision and at times diplopia for past thirty years. Staggers in the dark. Has unequal Argyll-Robertson pupils. Nerve shows atrophic disturbance. Some retinochoroidal changes. Impaired sensation in both legs. Sluggish patellar reflexes. In the Episcopal hospital fifteen years before with typhoid fever. Staggered in the dark and had shooting pains in legs at that time. Dr. G. O. Ring noted Argyll-Robertson pupils. Wassermann of serum negative; luetin, negative.

Spinal fluid: Cell-count, 15 lymphocytes; cell-count only made on spinal fluid in this case.

CASE 22.—C. B., aged 44. Tabes. Definite history of infection in 1893. Treated with mercury for one month in the secondary stage. First complained in February, 1912, of ataxia, failing vision, girdle sensation about the thorax and abdomen, pain and paresthesia in legs. Well nourished. Is unable to stand. Pupils irregular, react sluggishly to light. Complete optic atrophy. Subsiding neuroretinitis. Lower limbs ataxic, imperfect muscle sense, loss of patellar reflexes. Sphincter ani easily stretched. Priapism recently. He had been a painter but blood showed negative basophilia. Wassermann of serum negative on several occasions; luetin, negative.

Spinal fluid: Cell-count, 80 lymphocytes; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.2, 0.4, and 0.8 c.c., all dilutions negative.

CASE 23.—C. H., aged 49. Tabes. Denies infection. First complained of pain in left leg and side, nineteen years ago. Girdle sensation. Hyperphoria. Ataxia. Incontinence of urine. Is emaciated. Marked Romberg's. Ataxic, unable to walk. Unequal Argyll-Robertson pupils. Paralysis of third nerve. Has ophthalmoplegia externa. Ataxia of upper limbs. Loss of muscle sense in lower limbs. No patellar reflexes. Sphincter ani easily stretched. Wassermann of serum, 2 plus; luetin, negative.

Spinal fluid: Cell-count, 40 lymphocytes; Nonné and Noguchi, negative; Fehling's reduced; Wassermann, 0.2 c.c., negative, 0.4 c.c. 3 plus, 0.8 c.c. 4 plus.

CASE 24.—F. M., aged 55. Tabes. Charcot joint. Chancre thirty-four years ago. Treated with mercury for a short period. First complained of shooting pains in legs three years ago. Developed perforating ulcer of right foot. Charcot joint of left knee which became infected, was amputated above thigh. Has pinpoint pupils, no reaction to light. Has necrosis of end of left femur stump. Right patellar reflex absent. Sphincter ani relaxed. Wassermann of serum negative.

Spinal fluid: Cell-count, negative; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.5, 1.0 and 1.5 c.c. all dilutions negative.

CASE 25.—S. C., aged 37. Unilateral tabes. Denies all knowledge of primary or secondary lesion. Perfectly well till three and one-half years ago. Sole complaint since then irregular attacks of nausea and vomiting about a month apart. No apparent cause. No pain at any time. Anorexia during attacks. Unconscious of stomach at other times, and appetite then very good. Bowels, regular. Incontinence of urine at times, both day and night.

*Physical Examination.*—Eyes: right pupil dilated, rigid to light and accommodation; left uninvolved. Face: tremor of tongue and lips and right facial half subject to coarse contraction of muscles, involving malar region and wall of right naris. Very slight ataxia upper extremities. Legs: right knee-jerk absent, left, present. Both Achilles' reflexes absent. Slight ataxia. Painless perforating ulcer of left heel. Areas of diminished and delayed pain sense on trunk, ears and feet. Impairment of epicritic sense most marked about ankles. Romberg absent. Hearing unimpaired. Clinical diagnosis confirmed by Dr. Samuel D. Ingham, who believes also that early paresis cannot be ruled out. Gastric analysis was normal.

November 12, 1913: Wassermann of serum, 2 plus.

Spinal fluid: Cell-count, 5; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.2, negative; 0.4, negative.

November 18, 1913: Repeated because of the incomplete clinical picture. Wassermann of serum, 2 plus.

Spinal fluid: Cell-count, 10; Nonné and Noguchi, slightly positive; Fehling's reduced; Wassermann, 0.6 negative; 0.8, ½ plus; 1.0, 1 plus.

This shows very well the advantage of using larger amounts of spinal fluid. Spinal fluid each time under rather considerable pressure. Rectal examination at this time showed relaxation of sphincter ani, stretching causes no pain. No disturbance of sensation over skin of perineum or inner side of thighs.

December 26, 1913: Patient stated that clinical diagnoses of tabes in an early stage had been made two years prior. No laboratory work was done. One intramuscular of salvarsan given.

December 27, 1913: 0.6 of salvarsan intravenously.

January 3, 1914: 0.6 of salvarsan intravenously.

January 10, 1914: Began sodium cacodylate by hypodermatic injections, and mercuric chlorid by mouth. After 10 hypodermatic injections of sodium cacodylate, on February 5, 1914, 0.9 neosalvarsan intravenously.

February 16, 1914: 0.9 neosalvarsan intravenously and in all thirteen hypodermatics of sodium cacodylate, the majority 10 grains to the dose.

March 11, 1914: Wassermann of serum, 3 plus. Spinal fluid, not examined.

It will be noted that the Wassermann of serum has increased under four intravenous injections, which were really provocative. Subsequently used inunctions of 50 per cent. of mercurial ointment.

He is still subject to attacks of nausea and vomiting, though less severe; no longer feels as though walking on cushions of which he complained at the time of his first visit. All involuntary urination has ceased, both night and day. Pupils, reflexes about the same. Involuntary movements of the facial muscles less marked.

The result of the Abelin reaction in the urine after intravenous 0.6 gm. of salvarsan was negative after two, four and six hours; strongly positive after eight hours and negative after ten hours.

## TABETIC AND PARETIC CASES WHICH HAVE RECEIVED SUBDURAL TREATMENT, DIRECT OR SWIFT AND ELLIS

Case 26.—F. P., aged 40. Tabes. Lesion on penis in 1900, another one in 1903. The diagnoses of these were doubtful. Mercury taken for three months. No secondaries. First complaint was failing vision in 1904. At this time had Argyll-Robertson pupils, mixed treatment administered. In 1906 had gastric pains, for which at a later period he was operated on. For the past year is ataxic. Losing weight. Imperfect control over bladder and bowels. Sharp shooting pains in the legs. Is emaciated. Ataxic gait. Romberg's sign, positive. Pupils do not react to light or accommodation. Ataxic left leg. Patellar reflexes absent. Relaxed sphincter ani.

*Treatment.*—Swift and Ellis intraspinal treatment. Neosalvarsan 0.9 gm. was used for the intravenous injections. Forty per cent. of salvarsanized serum, heated to 56 degrees for half an hour, injected intraspinally.

*Reaction and Result.*—Severe pains in legs, epigastric pain and vomiting after each subdural. Less severe after second. After two injections—patient improved. Rapidly gaining in weight. Ataxia and urinary symptoms greatly improved. Feels subjectively better.

TABLE 1.—RESULT OF TREATMENT IN CASE 26[1]

| | Treatment | | |
|---|---|---|---|
| | Before | After | |
| | | First | Second |
| *Blood—* | | | |
| Luetin | — | | |
| Wassermann | 1+ | 1½+ | |
| *Spinal Fluid—* | | | |
| Cell-count: | | | |
| Lymphos | 30 | 25 | 10 |
| Polys | .. | .. | 20 |
| Nonné | ±? | ± | ± |
| Noguchi | ±? | ± | ± |
| Fehling's | ± | ± | ± |
| Wassermann in c.c. | 0.5, + | 0.5, + | 0.5, — |
| | 1.0, 2½+ | 1.0, 4+ | 1.0, — |
| | 1.5, 3½+ | 1.5, 4+ | 1.5,[2] |

CASE 27.—J. K., aged 43. Chancre in 1904. Treated with mercury for the following three months only. Complains of shooting pains and weakness in the legs, and ataxia, gradual in onset, in 1909. Poor vision. Imperfect control of urine. Is

emaciated. Marked ataxic gait. Marked Romberg's sign. Absorption of iris in both eyes. Double optic atrophy. Loss of patellar reflexes. No relaxation of sphincter ani.

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[1] In this and the following tables ++ = strongly positive, + = positive or Fehling's reduced, ± = slightly positive or slight reduction of Fehling's, ±? = very slightly positive.

[2] Less than 1+.

*Treatment.*—Two Swift and Ellis intraspinal treatments administered in the course of one month. Neosalvarsan 0.45 gm. for the intravenous injection, 40 per cent. salvarsanized serum injected intraspinally.

*Reaction and Result.*—Usual pains in the legs, more severe after first injection. Patient improved. Gait is less ataxic. Walks without the aid of a cane. Better control of urine.

TABLE 2.—RESULT OF TREATMENT IN CASE 27

| | Treatment | |
|---|---|---|
| | Before first | After first |
| *Blood*[1]— | | |
| Luetin[1] | + | + |
| Wassermann | 4+ | .. |
| *Spinal Fluid*— | | |
| Cell Count: | | |
| Lymphos | 20 | 60 |
| Polys | 20 | .. |
| Nonné | ± | — |
| Noguchi | ± | — |
| Fehling's | + | + |
| Wassermann, in c.c. | 0.2, — | 0.5, — |
| | 0.4, 1+ | 1.0, 3+ |
| | 0.8, 4+ | 1.5, 4+ |

CASE 28.—J. B., aged 61. Taboparesis. Denies infection. Pains in the legs and arms for the past three years. Now complains of ataxia, anesthesia and paresthesia of the hands and feet, girdle sensation. Imperfect control of the bladder and bowels. Diplopia. Is emaciated. Ataxic gait. Romberg's sign positive. Pupils are unequal, contracted, react to light but not to accommodation. Fundus shows beginning optic atrophy. Ataxia of upper limbs, fine tremor of the fingers of the left hand. Patellar reflex absent. Tremor of the left foot. Relaxed sphincter ani. Very advanced case. Bedridden for some time.

*Treatment.*—Swift and Ellis intraspinal treatment for four injections; 0.9 gm. of neosalvarsan for the intravenous injections; 40 per cent. salvarsanized serum heated to 56 degrees C. for one-half hour injected intraspinally.

*Reaction and Result.*—Sharp pains in the legs, vomiting. No improvement noted.

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[1] Luetin reaction—marked pustule, same reaction in negative control (*umstimmung Reaktion*).

TABLE 3.—RESULT OF ABELIN REACTION IN CASE 28

| | | Neosalvarsan | |
|---|---|---|---|
| | Hours | Intravenous | Intramuscular |
| Urine | 48 | .. | + |
| Blood | 1 | + | .. |
| | 2 | — | .. |
| Spinal fluid | 24 | — | .. |

TABLE 4.—RESULT OF TREATMENT IN CASE 28

| | Treatment | | | |
|---|---|---|---|---|
| | Before | After | | |
| | | Second | Third | Fourth |
| *Blood*[1]— | | | | |
| *Spinal Fluid*— | | | | |
| Cell count: | | | | |
| Lymphos | 70 | 5 | 30 | 70 |
| Polys | 3 | 3 | 20 | .. |
| Nonné | ± | .. | .. | ± |
| Noguchi | ± | .. | .. | ± |
| Fehling's | ± | .. | .. | + |
| Wassermann in c.c. | 0.1<br>0.2 } ++ | .. | .. | 0.2<br>0.4<br>0.8 } ++++ |

*Direct Injection and Final Result.* Two direct injections of 0.01 gm. of neosalvarsan suspended in 10 c.c. of normal salt solution. One intramuscular injection of 0.3 gm. neosalvarsan. No improvement. Marked symptoms of irritation. Less control of the bladder and bowels. Optic atrophy worse.

CASE 29.—G. M., aged 54. Tabes. Chancre thirty-four years ago. Mercury for a short time. Had moderate secondary symptoms. First symptom fifteen years ago, was shooting pains in the legs. Ataxic gait four years. Girdle sensation. Imperfect control over bladder and bowels. Is emaciated. Argyll-Robertson pupil. Marked ataxic gait. Marked Romberg's sign. Disturbed tactile and pain sense in the legs. Muscle sense absent. No patellar reflex. Relaxed sphincter ani.

*Treatment.*—Swift and Ellis, three weekly injections. Neosalvarsan 0.9 gm. for the intravenous injections; 40 per cent. salvarsanized serum intraspinally. Severe pain in the legs. No improvement was noted.

*Direct Injections.*—Two treatments, two weeks apart, of 0.015 and 0.01 gm. of neosalvarsan suspended in 12 c.c. of patient's serum and 18 c.c. of normal salt solution.

*Reaction and Final Result.*—Very severe pain in the legs, retention of urine for a short time. In addition, three intramuscular injections of 0.45 gm. neosalvarsan. Three intravenous injections of 0.9 gm. neosalvarsan. No improvement. Marked symptoms of irritation. Leg pains worse. Loss of control over bladder and bowels. Ataxia worse.

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[1] Wassermann $3\frac{1}{2}$ +, Luetin not tested.

TABLE 5.—RESULT OF ABELIN REACTION IN CASE 29

| | | Neosalvarsan | |
|---|---|---|---|
| | Hours | Intravenous | Intraspinal |
| Urine | 48 | .. | — |
| | 72 | .. | — |
| | 96 | .. | — |
| Blood | 1 | + | .. |
| Spinal fluid | 24 | — | .. |

TABLE 6.—RESULT OF TREATMENT IN CASE 29

| | Before | After | | | |
|---|---|---|---|---|---|
| | First | Second | Third | Fourth | Fifth |
| *Blood*— | | | | | |
| Luetin | — | .. | .. | .. | — |
| Wassermann | 3½+ | .. | .. | .. | 2+ |
| *Spinal Fluid*— | | | | | |
| Cell count: | | | | | |
| Lymphos | 180 | 60 | 44 | 12 | .. |
| Polys | 20 | .. | .. | 3 | .. |
| Endo | .. | .. | .. | 1 | .. |
| Nonné | — | .. | .. | .. | ± |
| Noguchi | ± | .. | .. | .. | ± |
| Fehling's | — | .. | .. | .. | + |
| Wassermann in c.c. | 0.2, — | .. | .. | .. | 0.2, — |
| | 0.4, — | .. | .. | .. | 0.4, — |
| | 0.8, — | .. | .. | .. | 0.8, — |

Case 30.—W. McN., aged 51., Chancre twenty-six years ago, no history of secondaries. First complained of anesthesia in the hands five years ago, which later involved the arms and legs. Now complains of ataxia, imperfect control of the bowels, shooting pains in the legs. Is emaciated, markedly ataxic gait, cannot walk or stand alone. Unequal pupils, responding sluggishly to light. Disturbed pain and tactile sensation and ataxia of both arms and legs. Absent reflexes of upper and lower extremities. Good tone to sphincter ani.

*Treatment and Result.*—Two Swift and Ellis intraspinal treatments. Otherwise treatment and reaction the same as the preceding case, that is, two direct injections. No improvement. Symptoms of irritation not so marked as in the preceding case. Optic atrophy appeared after treatment.

TABLE 7.—RESULT OF ABELIN REACTION IN CASE 30

| | Neosalvarsan | | |
|---|---|---|---|
| | Hours | Intravenous | Intraspinal |
| Urine . . . . . . | 48 | . . | — |
| | 72 | . . | — |
| | 96 | . . | — |
| Blood . . . . . . | $\frac{3}{4}$ | + | . . |
| | 1 | + | . . |
| Spinal fluid . . . . | 24 | — | . . |

TABLE 8.—RESULT OF TREATMENT IN CASE 30

| | Before | After | | |
|---|---|---|---|---|
| | | Second | Third | Fourth |
| *Blood—* | | | | |
| Luetin . . . . . . . | — | . . | . . | — |
| Wassermann . . . . | — | . . | . . | — |
| *Spinal Fluid—* | | | | |
| Cell count—Lymphos . | 60 | 36 | . . | . . |
| Nonné . . . . . . . | — | . . | . . | — |
| Noguchi . . . . . . | — | . . | . . | — |
| Fehling's . . . . . . | + | . . | . . | + |
| Wassermann in c.c. . . | [1] | . . | . . | 0.2, — |
| | . . | . . | . . | 0.4, — |
| | . . | . . | . . | 0.8, $1\frac{1}{2}$+ |

CASE 31.—J. K., aged 61. Incipient paresis. Chancre forty years ago. Mercury by mouth for four months. No secondaries. Has had within the last month three epileptiform seizures. Complains also of failing memory, constant headaches. Pupils negative. Slight unsteadiness on turning. Slight Romberg's sign. Patellar reflexes increased by reinforcement. Babinski suspicious, left toe. Tactile and pain sense not impaired.

*Treatment.*—Two direct injections of 0.01 gm. of neosalvarsan intraspinally, suspended in 12 c.c. of the patient's serum and 18 c.c. of normal salt solution. One direct injection of 0.02 gm. of neosalvarsan prepared in the same way.

*Reaction and Final Result.*—No noteworthy reaction following the injections. Has had two epileptiform attacks during the five months following the injections. Imperfect control over the bladder and bowels. Otherwise, no change.

The result of the Abelin reaction in Case 31 was negative in urine, before administration, negative in one, three, six nine and twelve hours afterward; positive in twenty-four hours, weakly positive in forty-eight hours and negative in seventy-two hours.

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[1] Anticomplementary substance interfered.

TABLE 9.—RESULT IN BLOOD AND SPINAL FLUID IN CASE 31

| | Treatment | | | |
|---|---|---|---|---|
| | Before | After | | |
| | | First | Second | 6 Mos. |
| *Blood—* | | | | |
| Luetin[1] | + | ... | ... | ... |
| Wassermann | 2+ | ... | ... | 2½+ |
| *Spinal Fluid—* | | | | |
| Cell count: | | | | |
| Lymphos | 250 | 300 | 180 | 40 |
| Polys | ... | 20 | ... | ... |
| Endo | ... | 20 | ... | ... |
| Nonné | — | ... | ... | + |
| Noguchi | ++ | ... | ... | + |
| Fehling's | ± | ... | ... | ±? |
| Wassermann in c.c. | 0.2, — | ... | ... | 0.5, — |
| | 0.4, ++ | ... | ... | 1.0, — |
| | ....... | ... | ... | 1.5, ± |

CASE 32.—W. S., incipient paresis. Denies infection. Epileptiform seizures for the past seven years. Nineteen attacks during the past year. No other complaint. General physical condition is good. Negative findings. Wassermann in blood, positive, +2.

*Treatment.*—One intravenous injection of 0.4 gm. of neosalvarsan. Three intramuscular injections of 0.3 gm. One direct intraspinal injection of 0.01 gm. of neosalvarsan, suspended in 12 c.c., of the patient's serum and 18 c.c. of N. S. S.

*Reaction and Final Result.*—Retention of urine for twenty-four hours occurring three days after the injections. No other reaction. Imperfect control of bladder and bowels. Epileptiform attacks still present.

The result of the Abelin reaction was negative in urine tested at intervals for three days.

CASE 33.—J. D., aged 47. Tabes. Sore on penis twenty-two years before. Disappeared in a few days. No knowledge of secondaries, though he had a sore throat which two physicians regarded as suspiciously luetic. Never took much medicine. Occasional sore throat since. Otherwise perfectly well till seven years ago. Then began to suffer from attacks of nausea and vomiting without pain, gradually increasing in severity and at intervals of shorter duration. Occasional fecal and urinary incontinence. Sensory disturbance, hypotonia, pupillary rigidity and myosis. Knee-jerks absent, moderate Romberg, paretic sphincter ani.

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[1] Luetin reaction delayed—papulopustular.

Such was his condition March 18, 1914. On two previous occasions, some months ago, he had been given salvarsan. March 22, 1914, the test of blood and spinal fluid resulted as follows: Wassermann in blood, positive; in spinal fluid in 0.5 c.c., 2+; 1.0 c.c., 4 +; 1.5 c.c., 4+. Cell count in spinal fluid, 30 lymphocytes; Nonné and Noguchi positive, Fehling's reduced.

March 28, 1914: 0.9 gm. neosalvarsan given intravenously.

March 29, 1914: 30 c.c., of 40 per cent. serum given subdurally.

March 30, 1914: Temperature rose to 101.8° F., but fell to normal next day, to remain down. Pains in legs—moderate.

April 3 and 4: Repetition of the above procedure. No temperature. Moderate pain in legs.

April 19, 1914: 0.006 gm. neosalvarsan given in 30 c.c. of physiologic salt solution, directly into subdural space. Sharp reaction in a few hours, very severe pains in legs, almost constant nausea, frequent vomiting, retention of urine, obstinate constipation. Catheterization and enemas necessary for three weeks. Absence of epigastric pain altogether with the presence of the autonomic reflex suggested gall-bladder as possible cause of the vomiting; referred to surgical service.

May 5, 1914: Operated on by Dr. W. Wayne Babcock. Gall-bladder thickened, densely adherent to omentum, no stones. Gall-bladder removed.

June 11, 1914: No relief from nausea and vomiting. Has had two attacks since operation. Knee-jerks have returned. Has control of bladder and bowel, though constipated. Romberg, absent. Pupillary phenomena the same. Greatly improved. Laboratory work not repeated.

Case 34.—A. J., aged 52. Paresis. History of infection twelve years ago. Been taking medicine practically ever since. Iritis about six years ago.

December 10, 1913: Homicidal and suicidal mania. Facial spasms. Speech thick, inarticulate at times. Suspicious, ungovernable appetite. Has insomnia. Lost memory. Cannot be trusted alone, no longer able to read. Unequal pupils, react to light and accommodation. Knee-jerks prompt. No Babinski or clonus. No relaxation of sphincter ani. Perfect control of bladder and bowel. Tremor.

Dec. 16, 1913, test was made on blood and spinal fluid.

TABLE 10.—RESULT OF TEST IN CASE 34

| | Treatment | |
|---|---|---|
| | Before[1] | After[2] |
| *Blood*— | | |
| Wassermann | +++ | +++ |
| *Spinal Fluid*— | | |
| Cell count—Lymphos | 20 | Normal |
| Nonné | ± | + |
| Noguchi | ± | + |
| Fehling's | + | + |
| Wassermann in c.c. | 0.2 — | 0.5, 4 + |
| | 0.4, 4 + | 1.0, 4 + |
| | 0.8, 4 + | 1.5, 4 + |

After December 16, 1913, he was given seven intravenous injections of 0.9 gm. neosalvarsan at weekly intervals, then three hypodermatic injections each week, each of sodium cacodylate, 15 grains.

*Result.*—Clinical improvement, none. Laboratory studies: Serum unchanged, +++. Spinal fluid unchanged as to Wassermann, cell-count fell to normal.

April 10, 1914: Subdural injection 30 c.c. of 40 per cent. neosalvarsanized serum. No reaction.

April 17, 1914: Subdural injection, direct, 0.008 of neosalvarsan in salt solution. Sharp reaction in a few hours, pain in hips and legs, vomiting which continued for two days, retention of urine necessitating use of catheter for twenty-four hours.

*Final Result.*—Gradual improvement. Mind now clear, memory good, reads paper and discusses topics, tremor almost nil, speech greatly improved. Pupillary phenomena unchanged.

We wish to take this opportunity of thanking Dr. Noguchi for the luetin which he kindly sent us and which we have used in over 100 cases of syphilis; also Dr. E. G. Alexander, chief of the Genito-Urinary Dispensary of the Episcopal Hospital, who permitted us to draw on his service for some of the cases studied.

[1] Test made December 16, 1913.

[2] Test made April 1, 1914.

### TABLE 11.—SUMMARY OF REACTIONS[1]

| Method of Neosalvarsan Injection | Patient | Case No. | Gm. | Urine[2] | Hours | Blood[3] | Hours |
|---|---|---|---|---|---|---|---|
| Intravenous | W. McN. | 30 | 0.9 | .. | ... | + | ¾ |
| | | 30 | 0.9 | .. | ... | + | 1 |
| | G. M. | 29 | 0.9 | .. | ... | + | 1 |
| | M. S. | 2 | 0.45 | + | 3½ | + | ¼ |
| | | 2 | 0.45 | ± | 5½ | + | ½ |
| | | 2 | 0.45 | — | 8 | ± | 1 |
| | | .. | ... | .. | ... | — | 1½ |
| | J. K. | 27 | 0.45 | .. | ... | + | ½ |
| | | 27 | 0.45 | .. | ... | — | 2 |
| | J. B. | 28 | 0.9 | .. | ... | + | 1 |
| | | 28 | 0.9 | .. | ... | — | 2 |
| | P. F. | 1 | 0.9 | .. | ... | + | ¼ |
| | | 1 | 0.9 | .. | ... | + | ½ |
| | | 1 | 0.9 | .. | ... | — | 1 |
| | | 1 | 0.9 | .. | ... | — | 1½ |
| | S. C. | 25 | 0.6 | — | 2 | — | 1 |
| | | 25 | 0.6 | — | 4 | — | 1½ |
| | | 25 | 0.6 | — | 6 | .. | .. |
| | | 25 | 0.6 | ++ | 8 | .. | .. |
| | | 25 | 0.6 | — | 10 | .. | .. |
| Intramuscular | M. S. | 2 | 0.3 | ± | 1 | .. | .. |
| | | 2 | 0.3 | + | 3 | .. | .. |
| | | 2 | 0.3 | ± | 5 | .. | .. |
| | | 2 | 0.3 | + | 8 | .. | .. |
| | | 2 | 0.3 | + | 12 | .. | .. |
| | | 2 | 0.3 | + | 24 | .. | .. |
| | | 2 | 0.3 | ± | 31 | .. | .. |
| | | 2 | 0.3 | — | 33 | .. | .. |
| | J. B. | 28 | 0.3 | + | 48 | .. | .. |
| Intraspinal | W. Mc.N.[4] | 30 | 0.01 | — | 24 | .. | .. |
| | | 30 | 0.01 | — | 48 | .. | .. |
| | | 30 | 0.01 | — | 72 | .. | .. |
| | G. M. | 29 | 0.015 | — | 24 | .. | .. |
| | | 29 | 0.015 | — | 48 | .. | .. |
| | | 29 | 0.015 | — | 72 | .. | .. |
| | W. S. | 32 | 0.01 | — | 3 | .. | .. |
| | | 32 | 0.01 | — | 8 | .. | .. |
| | | 32 | 0.01 | — | 12 | .. | .. |
| | | 32 | 0.01 | — | 24 | .. | .. |
| | | 32 | 0.01 | — | 36 | .. | .. |
| | | 32 | 0.01 | — | 48 | .. | .. |
| | | 32 | 0.01 | — | 72 | .. | .. |
| | J. K. | 31 | 0.01 | — | 1 | .. | .. |
| | | 31 | 0.01 | — | 3 | .. | .. |
| | | 31 | 0.01 | — | 6 | .. | .. |
| | | 31 | 0.01 | — | 9 | .. | .. |
| | | 31 | 0.01 | — | 12 | .. | .. |
| | | 31 | 0.01 | + | 24 | .. | .. |
| | | 31 | 0.01 | ± | 48 | .. | .. |
| | | 31 | 0.01 | — | 72 | .. | .. |
| | | 31 | 0.02 | — | 3 | .. | .. |
| | | 31 | 0.02 | — | 8 | .. | .. |
| | | 31 | 0.02 | + | 24 | .. | .. |
| | | 31 | 0.02 | — | 48 | .. | .. |
| | | 31 | 0.02 | — | 72 | .. | .. |

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[1] Spinal fluid tested only in cases of W. McN. and G. M. Result, negative after twenty-four hours.

[2] Control, specimen of urine voided just before the injection of neosalvarsan was tested.

[3] Control, specimen of serum free of neosalvarsan was tested. An unsuccessful attempt made by means of the Abelin reaction to determine whether any of the neosalvarsan was taken up by the erythrocytes or whether all of it goes into the serum.

[4] Arsenic found in urine after subdural injection.

# CONGENITAL SYPHILIS, WITH REPORT OF A CASE

By JOHN PAUL JONES, M.D.
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Syphilis has been known to the profession as a morbid entity since the time of the siege of Naples by Charles VIII (1494-1495), but it has been only during the past few years that remarkable strides have been made in its diagnosis and treatment, both of which have become comparatively easy in the primary and secondary stages, and in most cases of the tertiary. But the long-neglected undiagnosed and untreated congenital syphilitics who present hideous deformities of almost every tissue and organ of the body, are subjects whom many of us are not anxious to treat, and whom we had much rather see go to someone else. But these individuals offer a field for patient work that is usually rewarded with splendid results. Badly damaged organs cannot be made normal, but such organs often can be made to functionate in a manner consistent with comfort and health, greatly to the patient's relief and the physician's satisfaction.

I report the following case not because there is anything especially unusual about it except its severe nature, but chiefly to impress the fact that patient treatment can do untold good to many of these most miserable of all people.

Lillian W., aged eighteen years, was admitted to Dr. Ashhurst's orthopedic service in this hospital, August 19, 1913.

*Family History:* Father died sixteen years ago of heart disease, which was most probably syphilitic, for as Stoll says sudden cardiac death occurring in middle life is almost always syphilitic

("Role of Syphilis in Hypertensive Cardiovascular Disease," H. F. Stoll, *Albany Med. Jour.*, August 1915). Mother died four years ago of cancer of the stomach (?). The first child lived for two years. This patient is the second child. Five other children followed, but none of them lived more than two days.

*Personal History.* There is nothing of moment in the patient's personal history until the age of seven years. Prior to this her health had been good, except some of the acute exanthemata common to childhood. She was probably a little large for her age. At the age of seven she accidentally ran a piece of glass into her nose just below the left eye; the glass was removed by a physician. About nine months after this accident her mother noticed that the child's nose was beginning to sink in slightly at the bridge. Three months later the entire septum of the nose had disappeared, thus permitting her nose to lie flat.

At the age of ten years she was four feet nine inches in height. She began menstruating at the age of thirteen, and this function has always been regular, occurring every twenty-eight days and lasting two days. During her thirteenth year she became bedridden owing to the following occurrences: her right leg in one night became flexed at the knee, and has remained in this condition since. However, previous to this time, she had had pain in the knee-joint, and she states she would at times hear a grating in the joint on flexion and extension. About nine months after the occurrence of this fixed flexion of the limb, she fell and sustained a greenstick fracture of the right leg. Three years later (two years before admission) the left knee became flexed in a manner similar to the right; this occurred during one night also. She states she had not previously had any pain in the left knee, as she had had in the right. About the same time the right forearm became slightly flexed at the elbow, and she has been unable to extend it completely since. One year ago the patient attempted to straighten out the left lower extremity and as a result of this she fractured the femur about its middle. This fracture united with an anterolateral convex deformity (Fig. 2), and one week before admission she refractured it, coming into the hospital with no union. About one year ago the middle of the right forearm became markedly bent with the convexity on the flexor surface (Fig. 3), the bones forming an angle of about 120 degrees.

For the past *five years* this girl had not once been removed from a wheel chair. In this she spent her days and slept at night, used the bedpan when necessary, and had the pillows and coverings changed as occasion demanded, but with great difficulty and suffering. She lived with her aunt, all the members of her immediate family having perished from the disease which was the only legacy she herself had received from them. She spent her life in a little room back of a grocery shop, kept by her aunt, and seldom or never saw any outsiders.

*On admission* the patient is seen to be anemic, has a very pasty appearance, and is exceedingly hypersensitive, emitting the most frightful screams at the slightest attempt to turn her in bed or bathe her. She is entirely unable to move either of her lower extremities, and she has only limited motion in her upper extremities. She has to be fed, and is in every way absolutely helpless. She is an excessively miserable-looking creature.[1]

Her hair is light in color, and there is no evidence of alopecia, The eyebrows are thin, and there is an entire absence of pubic and axillary hair. The pupils are equal and react normally to light and accomodation. Over the left lachrymal duct there is a swelling that apparently contains pus. The bridge of the nose is present, but the upper part of the alae and the tip of the nose have ulcerated away. A great part of the septum is missing, and the under part of the nasal bones is eroded together with the soft parts underlying them. There is a continuous mucopurulent discharge from the nose (examination of this by Dr. C. Y. White showed no evidence of Treponema pallidum).

The tongue is normal. The teeth are not typical "Hutchinson" in type; they are normal in size, but the two upper incisors are very loose and movable. The roof of the mouth is normal, with the exception of a perforation of the soft palate.

The chest is flattened laterally, projecting in front (pigeon breast). Expansion is fair, but the tactile and vocal fremitus are increased over the right chest posteriorly.

The heart is apparently normal. The abdomen is slightly protruding, and the liver extends to within about one inch of the umbilicus. The spleen is slightly enlarged.

The muscles about the right shoulder-joint are more or less atrophied, but the joint is freely movable. The right forearm is held in acute flexion with elastic resistance to extension. The bones of the forearm are very elastic and weak, scarcely being able to support the weight of the hand. There is marked anterior angularity of the mid-forearm, the forearm is very tender, the patient screaming at the least manipulation. The proximal phalanx of each finger presents a marked spindle-shaped hypertrohy (typical syphilitic dactylitis).

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[1] It is worth while to record the fact that she was at this time so repulsive to look at that one of the resident physicians who went to look at her, as a pathological curiosity, became nauseated at the sight, and that one of the nurses in training actually vomited after getting one good look at her.

The left upper extremity is practically the same as the right except there is not so great angularity in the forearm; the deformity suggests an old Colles's fracture. The left lower extremity presents a fracture of the femur about the middle third; the fragments can easily be felt, and their movements seen beneath the skin on the anterior surface of the thigh. The leg, with the lower femoral fragment, is in marked internal rotation on the upper fragment. There is no union. The left knee is in a position of fixed flexion of about 90 degrees. The left leg is very flexible at its lower third, with very little of the tibia present. The dorsal surface of the foot is swollen, and the foot is in a position of marked valgus. The toenails are loose, and pus pours from beneath them.

The right lower extremity presents a flexion at the knee of about 90 degrees. The knee is not ankylosed, but very little movement is possible. The lower third of the leg is very flexible indeed, and the tibia cannot be felt at all at this level, and skiagraphs show its continuity lost (Fig. 4). When the calf of the leg is lifted from the bed, the lower third of the leg drops to a position at right angles with the upper portion. The foot is large and pudgy, and is in considerable equinovarus deformity. The patient sits or reclines in bed (she cannot lie down) with the right leg drawn under her and the left knee supported on the right one.

Immediately after her admission a Wassermann reaction was made on her blood serum, and was found to be positive *plus four*. She was put on protiodide of mercury and potassium iodide, but these had to be discontinued in about two weeks owing to soreness of her gums. Scrupulous cleanliness was observed, and she was given a special diet of the most appetizing food. She was regularly given general massage, and was kept on the porch day and night in all weathers.

August 23, 1913. Patient is given 0.4 gm. of neosalvarsan, which caused nausea for four or five hours. Her thighs are being gradually separated by interposition of pillows.

September 20. Is given another injection of 0.4 gm. of neosalvarsan; this was followed by no reaction. The abscess of the lachrymal duct was incised and dressed.

Injections of neosalvarsan were continued about every two weeks, and by October 10, the patient seems to be improved in many ways. Her color is much better. Her teeth are not so loose. Both forearms have a much wider range of motion. The bones of the forearms and legs seem to be thickening; they are certainly much stronger, and she can be moved about in bed without exciting such great pain. The sinus in the lachrymal duct does not improve very much, under repeated dressings, and the purulent exudate from beneath the toenails does not improve.

October 20. The discharge about the toes has ceased and the patient can lift up her entire right lower extremity without pain.

November 13. Ulceration of the left eye has almost healed. An adhesive plaster extension apparatus is attached to the left knee, and weights are hung to it over the side of the bed, in an effort to produce abduction of the left thigh. The girl is now able to feed herself.

November 17. The extension on the left knee has produced marked lessening in the adduction. The patient is now given one grain of salicylate of mercury in oil, by intramuscular injection, every two weeks. She can flex and extend the right leg on the thigh but cannot extend it beyond a right angle. She is evidently gaining in weight. Her appetite is fair, and she does not complain of pain while she is being bathed and moved about in bed. She continues to give a plus 4 serum Wassermann.

November 20. The right lower extremity is put on a right-angle splint, like a double inclined plane, and this is held perpendicular to the bed; but attempts to extend the leg on the thigh beyond a right angle produce a marked bend of the leg a few inches below the knee instead of extending the knee joint (Fig. 2).

November 23. The splint to the right lower extremity holds the leg in very good position, correcting the bend below the knee. The left thigh has now been abducted until it lies in the long axis of the body.

November 30. Some soreness of the gums and some abdominal pain, so the mercury injections had to be discontinued for the time being.

January 1, 1914. The salicylate of mercury treatment has been given again for some time without any ill effects. Her general appearance has been greatly improved. The ulcerating area at the inner angle of the left eye has healed. The right knee can now be extended to 105 degrees (15 degrees more than a right angle).

January 20. The patient was placed in a chair today with her feet in another chair. She sat up for several hours in perfect comfort.

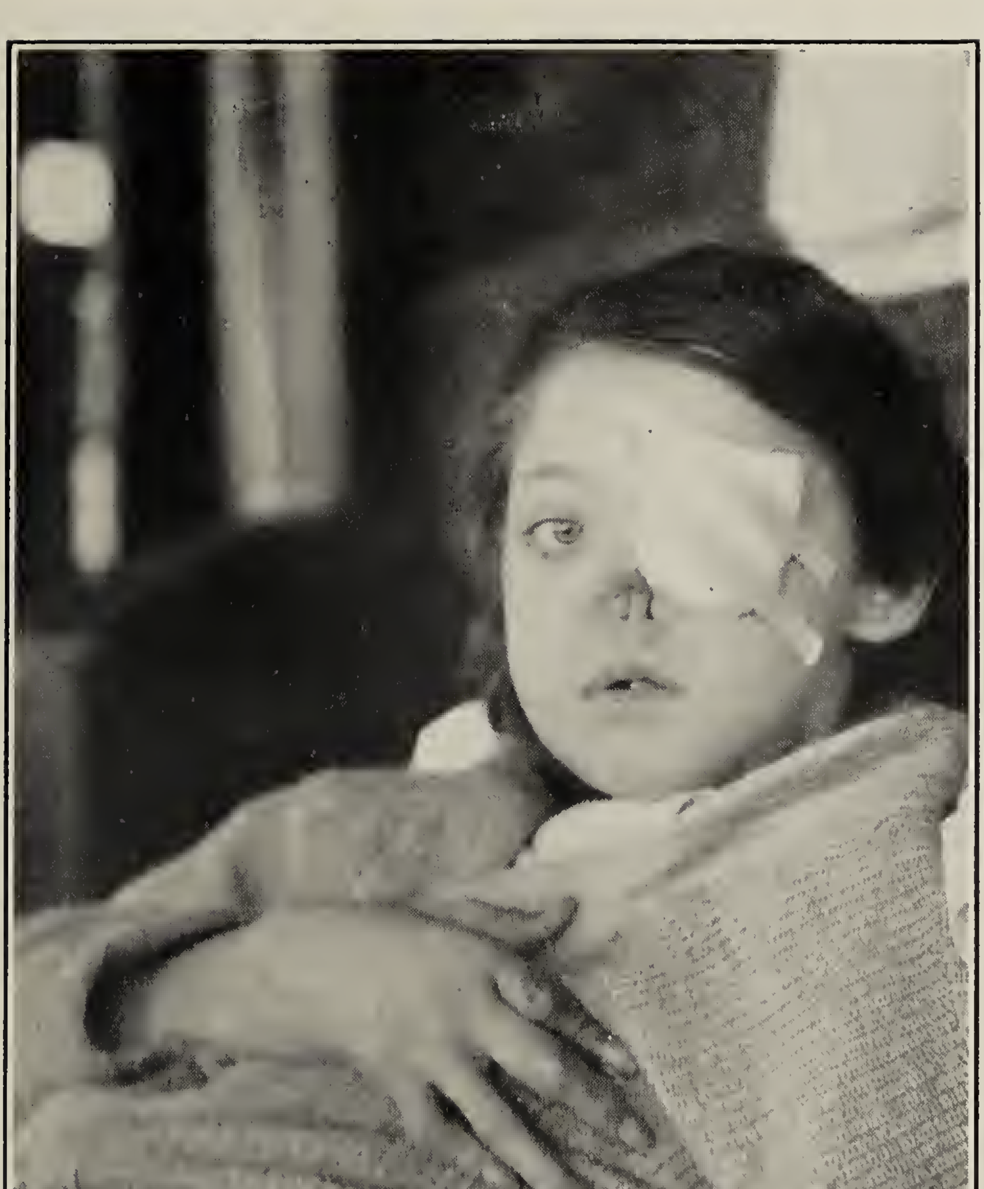

FIG. 1.—Typical syphilitic dactylitis.

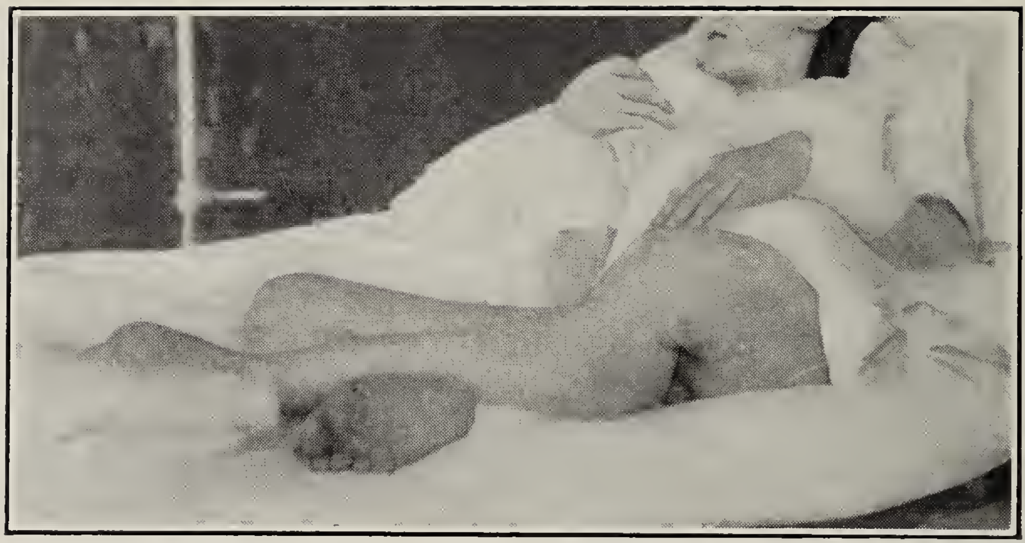

FIG. 2.—Pathological fracture of left femur; note deformity of legs and foot.

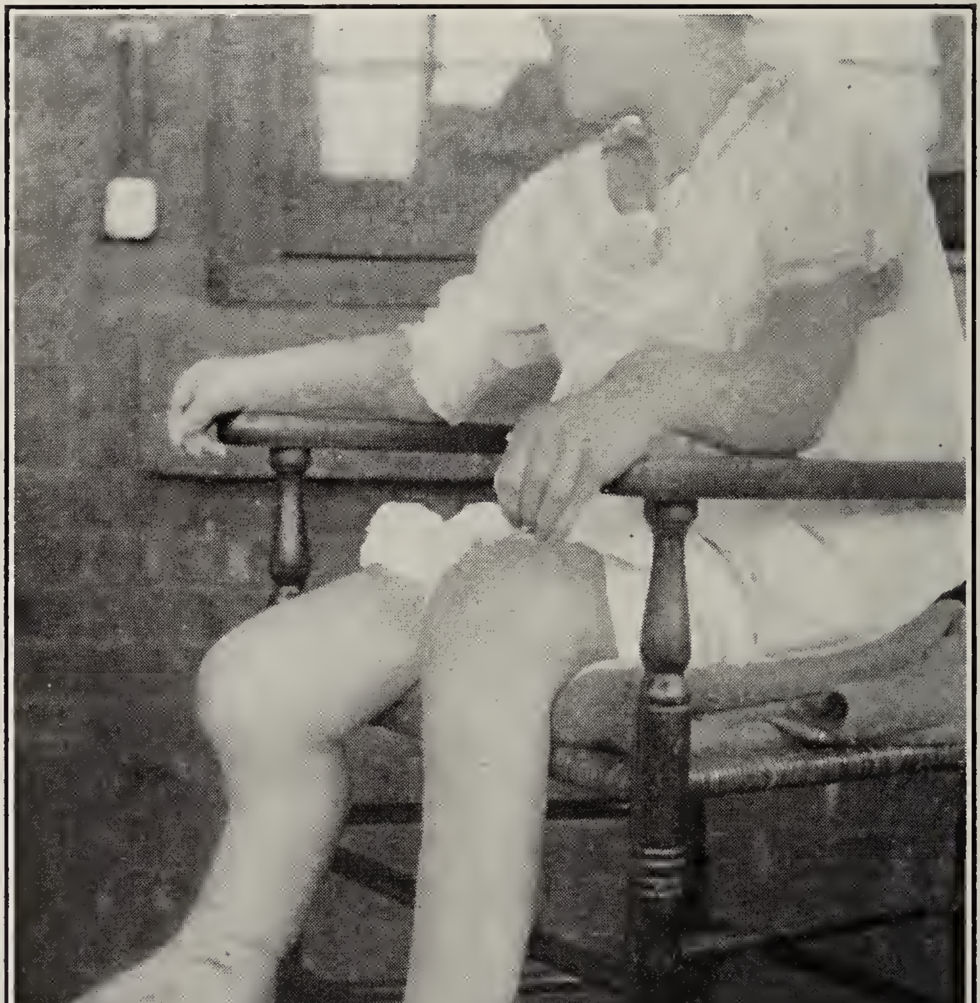

FIG. 3.—Showing deformity of arms and right leg.

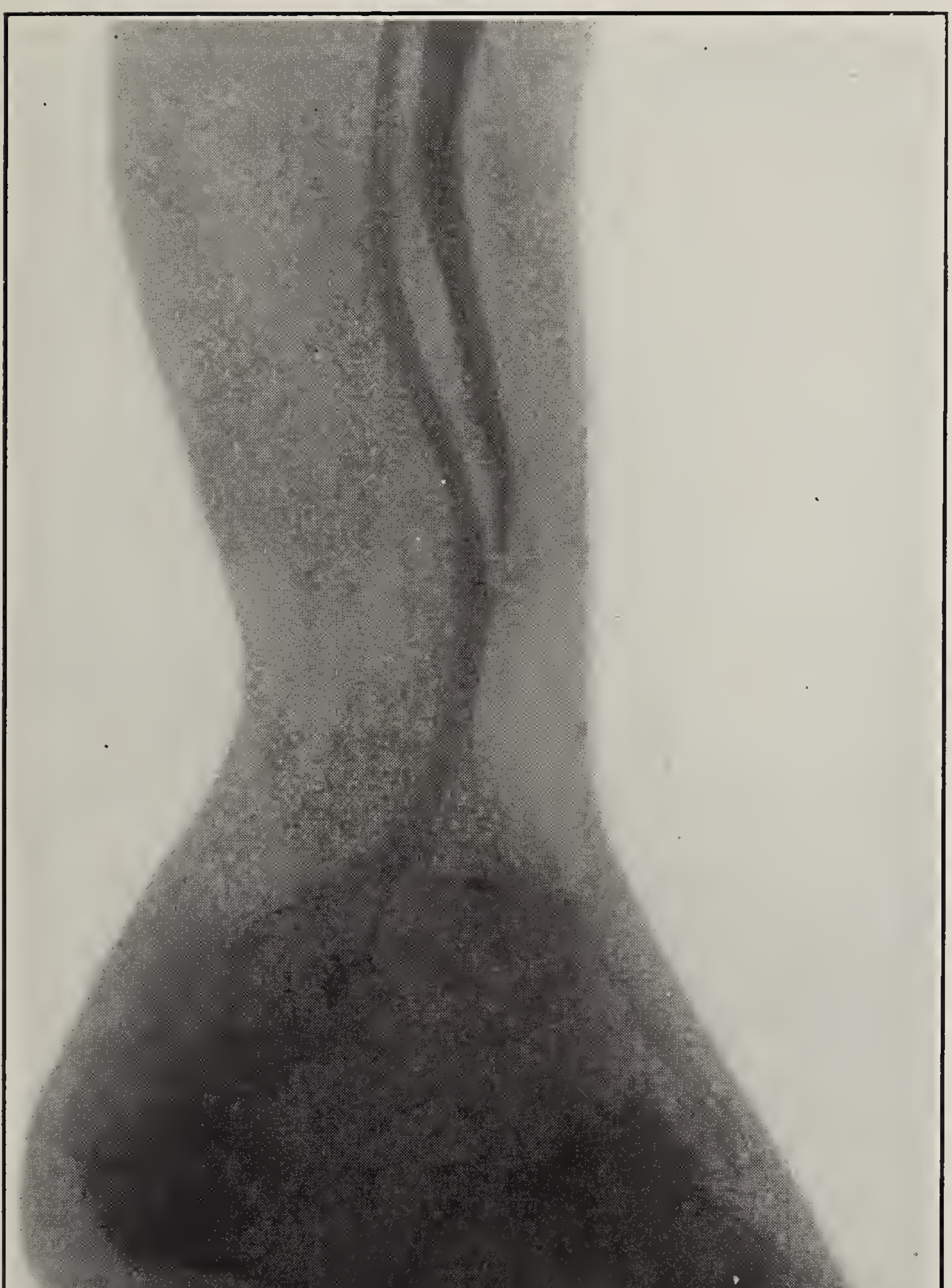

FIG. 4.—Right leg; extreme bone atrophy, occurring in hereditary syphilis; the continuity of the tibia is lost, that of the fibula present.

March 17. Luetin test was done two days ago by Dr. J. V. Klauder. Today it shows an area of erythema about 2 cm. in diameter, a markedly inflamed papule at its centre, with slight induration surrounding it. She is now given one grain of salicylate of mercury intramuscularly every two or three weeks. At times the intervals have to be lengthened owing to soreness of the gums. The Wassermann blood serum reaction continues to be plus 4.

June 5. The patient sits in a chair all the day now and seems to be feeling perfectly well. Braces were applied to her forearms today in the hope that they might be somewhat straightened.

July 8. General condition is greatly improved. She is discharged and referred to the orthopedic dispensary for further treatment. She is instructed to continue her mixed treatment as in the hospital, returning at suitable intervals for the mercury injections.

January 7, 1915. The patient is readmitted to the hospital today. The sinus below the inner canthus of her left eye has opened again. She is now able to get in and out of her wheel chair and into and out of bed alone, but she cannot stand even with support. She has no pain in any of her joints. While at home she tried to stand and fell, breaking her right leg above the ankle; she kept it on a splint for three weeks, when it had apparently united firmly. (This is the leg in which the tibia had become so atrophic that at one portion it did not cast any shadow in the *x*-ray. (See Fig. 4, made August 20, 1913). She can use her hands almost as well as a normal person; she sews, embroiders, and writes a legible hand. She can extend her right elbow to 125 degrees and flex it to 35 degrees. The right forearm at the middle shows an angularity of 120 degrees with the apex on the flexor surface. Wrist motion is nearly normal. The left elbow can be extended to 135 degrees and flexed to 35 degrees. There is subluxation forward of the left wrist, with some thickening of the lower end of the radius and deviation of the hand to the ulnar side. Rotation in each forearm is about half normal. The fingers are nearly normal.

She can extend her right knee to 135 degrees and flex it to 55 degrees. The bones of the right leg are bent backward in the upper third to an angle of 140 degrees, with the apex on the calf surface. the tibia and fibula are tender above the ankle (site of the recent fracture). The right foot is in a position of equinovarus. The ankle can be flexed to 120 and extended to 130 degrees. The foot is large and pudgy. The toes are nearly normal.

The left knee can be extended to 120 degrees and flexed to 85 degrees. The left leg is straight. The foot is in considerable valgus deformity. The flexion in the ankle joint is 90 degrees, and extension to 105 degrees.

The liver and spleen seem smaller than when she first came under treatment 16 months ago.

On readmission into the hospital she continued her old treatment of one grain of mercury salicylate intramuscularly every two weeks, with potassium iodide in increasing doses until iodism, then discontinuing for two or three weeks.

January 30. Wassermann serum reaction plus 2.

March 10. Wassermann serum reaction plus 2.

April 28. On this date Dr. Ashhurst performed manual osteoclasis of the right radius and ulna, under gas-oxygen anesthesia. Union was fair and position good by the middle of June, when she was discharged for another vacation.

October 20. The patient has been admitted into the hospital again, for the purpose of osteoclasis of the left forearm, which was done by Dr. Ashhurst on this date. Union and position good.

January 23, 1916. The patient, who has been home since the last operation, writes asking to be readmitted, and inquiring about operations on her lower extremities, and for the formation of a nose.

While we have not obtained either an anatomical or laboratory cure in this case still our results have been gratifying and have well repaid us for our efforts. In less than two years this breathing piece of pathology has been changed, by rather simple treatment, from a miserable, loathsome, deformed, ulcerating, hypersensitive, screaming wretch to a much less deformed, happy, fairly healthy person, who can use her mind and her hands in various methods of interesting and profitable work. The most interesting part of the surgical work required could not be done until this stage was reached. It now remains to overcome the deformities of the lower limbs by a series of more or less complicated operations, and to teach her to walk. Also to form a new nose. Her own changed attitude to life is evidenced by the fact that when she came under treatment first, she said the absence of her nose was "the least of her troubles." Now she regards it as almost the greatest.

# THE VALUE OF FUNCTIONAL KIDNEY TESTS IN MEDICAL CONDITIONS*

By GEORGE MORRIS PIERSOL, M.D.
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During the last few years tests for estimating renal function have received careful attention, and useful data have been accumulated. All observers along these lines have endeavored to devise methods by which (1) nephritis may be recognized before the usual clinical signs become manifest; (2) the exact variety of nephritis may be determined, *i. e.*, whether the lesions are chiefly glomerular, tubular, or both; and (3) the outcome and duration of the case may be predicted.

The first clinical and experimental work to throw much light on renal function was that of Schlayer and his associates[1] in 1910. They produced experimental nephritis of various types in rabbits by potassium bichromate, cantharides, and uranium nitrate. In this way they were enabled to observe the effect of various substances such as water, salt, lactose and potassium iodide in vascular, tubular and mixed types of kidney lesions. Later similar observations were carried out on patients suffering from various clinical types of nephritis. The result of Schlayer's extensive clinical and experimental observations may be summed up as follows:

*Vascular nephritis*, both acute and chronic, may be recognized by delayed lactose excretion, by oliguria and polyuria depending upon whether or not the renal vessels are sensitive or insensitive to vasodilator influences, and the normal excretion of iodides and chlorides.

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* Read at a meeting of the Episcopal Hospital Clinical Society, March 16, 1914.

*Tubular nephritis*, on the contrary, is characterized by delayed chloride and iodide excretions, with a normal water and lactose output.

Subsequent experiments by numerous observers, have in the main confirmed these findings, although there are some investigations that are at variance with Schlayer's results. The criticism has been made by Christian[2] that in too few of the cases thus examined functionally, have the findings been correlated with subsequent histological examination of the kidneys to justify as yet complete acceptance of the significance of these tests. There seems little doubt but that the functional tests of Schlayer do make possible the early recognition of abnormalities of renal function and are capable of indicating the progress of early kidney lesions. It is questionable, however, whether these tests are as useful in differentiating clinically the vascular and tubular types of nephritis, as they are in experimental renal disease. It is held that these tests are of less value in advanced kidney disease than in the earlier stages, since in all well-developed forms of nephritis the lesions are diffuse, and wide-spread changes co-exist in both tubules and glomeruli.

In all forms of nephritis, both acute and chronic, certain substances are retained, accumulate in the blood, and are presumably the cause of the toxic symptoms that arise. As an aid in determining the degree of functional incapacity that exists in advanced nephritis, it has been shown to be of importance to detect such retention and to estimate its degree. This may be accomplished by determining the freezing point of serum (cryoscopy) and by quantitatively determining the total incoagulable nitrogen and urea in the blood. The simplified methods recently devised by Folin and Denis[3] for the determination of total incoagulable nitrogen, and by Marshall[4] for urea, have made it possible to perform these estimations in any well-equipped laboratory; they necessitate the use of only small quantities of blood, 2 to 5 c.c. as against the large quantities formerly required by the older, more complicated methods.

It has been shown that an increased concentration of total incoagulable nitrogen and urea in the blood does not occur in all cases of severe renal disease, therefore normal amounts of these substances in the blood do not invariably mean normal kidneys. A noteworthy increase, however, of these substances in the blood does point to serious renal involvement and generally speaking, the greater the increase of these substances in the blood, the greater the degree of renal injury. Urea concentration of more than 0.55 gm. and total incoagulable nitrogen in excess of 0.5 gm. per liter, together with a serum freezing point lower than –60° C. are regarded by Rowntree[5] as of great prognostic significance and such evidences of retention are termed by him "cumulative phenomena."

The above tests for the recognition of cumulative phenomena, though relatively simple, are obviously inapplicable to many cases and must of necessity be beyond the reach of the ordinary clinician. Happily, however, there has been devised a simple excretory test by which, *total* renal function may be determined and which, therefore, is useful in all forms of nephritis whether early or advanced, namely, the phenolsulphonephthalein test of Rowntree and Geraghty,[6] a test which has rightly been described by Fitz[7] as "the most valuable single aid to the estimation of *total renal* function which is known at present." By much painstaking study the authors of this test have adequately demonstrated that the quantity of phenolsulphonephthalein eliminated by the kidneys in a given time, varies in direct proportion to the degree and severity of renal involvement.

The test is easily and quickly carried out, is accurate and is devoid of all possible harm to the patient. It depends upon the fact that 'phthalein is a dye which is eliminated practically entirely through the kidneys and is absolutely non-toxic.

Although the point is not settled, it is probable that in the main, phenolsulphonephthalein is largely excreted by the tubules. This action, however, is not sufficiently selective to make it possible to determine accurately by means of 'phthalein excretion the pathological type of nephritis, as may possibly be done in early cases by Schlayer's tests. 'Phthalein is useful as a means of recognizing an impairment of *total* renal function.

This 'phthalein test has been extensively employed during the past year on my medical service at the Episcopal Hospital in all cases in which diminished renal function was suspected. The technique of the test as employed was: A solution of 6 mg. of 'phthalein to 1 c.c. of salt solution was used. This was injected intramuscularly. All the urine secreted was then collected at the end of one hour and again at the end of the second hour. The urine was either collected by catheter or was voided, the latter method being usually used. The urine was then rendered alkaline by 2 per cent. sodium hydroxide solution, and the per cent. of 'phthalein estimated by comparing specimens of the urine with the standardized tube of the Dubosc colorimeter.

Under normal conditions 40–60 per cent. should be recovered in one hour and 60–80 per cent. at the end of two hours. In our experience even in apparently normal individuals, the readings are not as a rule as high as these.

The excreting capacity of the kidneys has also been tested by the use of other dyes, such as indigocarmine, rosaline, and methylene blue, but none of these substances has been found as generally useful or accurate as has phenolsulphonephthalein.

Although the 'phthalein test has been largely used by numerous observers, and reports as to its value are generally favorable, it may be of some profit to consider the results obtained from the application of this functional test to medical cases. The influence of the test is perhaps best shown by briefly citing cases illustrative of certain types of diseases in which the kidneys were more or less involved.

As might be expected, the most marked impairment of 'phthalein excretion was encountered in acute uremia, indeed in some cases this deficiency amounted to absolute suppression.

This was demonstrated in the case of a woman, aged thirty-one years, who was admitted in a semistuporous condition. Her systolic blood-pressure was 200, diastolic 145, the heart was hypertrophied, and the urine contained a cloud of albumin, but no casts. It did, however, contain some acetone and diacetic acid. There was absolutely no excretion of 'phthalein. Six weeks before admission she had complained of some dyspnea and edema of the legs. Four days before admission she had had a convulsion followed by unconsciousness for three hours and by a slight hemiplegia. A few hours after coming to the hospital she became comatose, had several general convulsions, and died.

This case is of interest because of the acidosis that was present in view of the fact that some observers have held that certain of the toxic symptoms of uremia are the result of acidosis.

The prognostic value of the test was well shown in a case of delirium tremens occurring in a middle-aged man.

When first admitted he showed no noteworthy urinary findings except a cloud of albumin and a few hyaline casts and the urinary output was normal. Several days later, although his delirium tremens was better and the urinary condition unchanged, the 'phthalein test showed that no dye was eliminated. Repeated tests confirmed this observation, and an unfavorable prognosis was given. One week later although the man seemed improved, the urinary output lessened, more albumin appeared in the urine, the 'phthalein test continued to show an absence of all dye output and the patient promptly succumbed in uremic coma.

Another fatal case of uremia observed in a woman, aged forty-two years, gave surprisingly opposite findings.

Two days before admission she had been seized with vertigo followed by a general convulsion. When brought to the hospital she was exceedingly excitable. There were no palsies. The day after admission she had a slight convulsion followed by marked drowsiness; the following day she had a general convulsion from which she lapsed into coma, followed by death. Her urine contained a trace of albumin and a few hyaline casts. Her blood-pressure was systolic 145, diastolic 110. A lumbar puncture revealed bloody cerebrospinal fluid under considerable tension. Her heart was not much hypertrophied. Her 'phthalein excretion, however, was 69 per cent. for two hours, although at autopsy, in addition to a cerebral hemorrhage, the marked red, granular, contracted kidneys of chronic interstitial nephritis were found.

Pathologically there was no doubt as to the existence of chronic interstitial nephritis, yet the 'phthalein excretion was about up to normal. This case resembles somewhat the case reported by Austin and Pepper[8] in which a nephritis associated with edema existed, but in which the 'phthalein was 67 per cent. for one hour and total incoagulable nitrogen normal, but the chloride output was somewhat delayed. Baetjer[9] has more recently reported four similar cases. In the above case the entire absence of edema suggested that the chloride output was probably normal, but the absence of any tests save that for 'phthalein secretion makes it impossible to arrive at any accurate idea as to the functional state of the kidneys. The existence of cases of marked nephritis with kidneys hyperpermeable to 'phthalein must not be lost sight of.

The majority of cases of chronic nephritis of all kinds showed in addition to the usual urinary and cardiovascular phenomena, a persistently low 'phthalein output throughout the entire period of observation regardless of whether the cases ended unfavorably or improved. For example, in one, a man aged sixty-six years, who died as the result of a hemorrhage incidental to a coexisting portal cirrhosis, the 'phthalein at the end of two hours always ranged from 23 per cent. to 15 per cent. In a man, aged fifty-two years who had undoubted chronic interstitial nephritis with a blood-pressure of systolic 185, diastolic 105, in spite of marked subjective improvement, 'phthalein excretion was 27 to 31 per cent.

Several individuals of middle age or over have come under observation who, in spite of definite clinical signs of chronic nephritis, have consistently shown a 'phthalein excretion almost up to the normal. In one at the end of two hours it was 48 per cent; in another 50 per cent.; in a third, 64 per cent.; and in a fourth, 52 per cent. Such cases as these make it evident that no one test of renal efficiency should be depended upon solely, and that it is impossible to diagnosticate the presence of nephritis from the 'phthalein test alone. Unless it be associated with a careful general examination and other clinical data, the 'phthalein test is of little value. In these cases of clinical nephritis with good 'phthalein output, other functional tests such as the estimation of blood urea and total non-coagulable nitrogen must also be performed if we hope to arrive at an accurate conception of the status of the renal function.

A group of cases frequently encountered, generally termed cardiorenal, in which a variety of cardiac, vascular, and renal conditions coexist, often show when they first come under observation, alarmingly low 'phthalein excretion. Under suitable treatment not infrequently the urinary findings improve and coincidently the 'phthalein output increases up to or almost to normal. In such cases the renal condition is largely secondary to the condition of the circulation. Such a case was observed in a man, aged fifty-eight years, who was admitted complaining of dyspnea and edema. His heart sounds were poor and his heart somewhat dilated, and the urine contained a little albumin. The first 'phthalein test showed only a faint trace of the dye at the end of two hours. Twenty days later, the patient having experienced marked subjective improvement, the 'phthalein excretion had risen to 31 per cent. in two hours. In these cases the test is of value as a means of determining the degree of actual kidney involvement and how far the cardiac insufficiency is responsible for the conditions present.

In acute nephritis this functional kidney test shows a fairly consistent decrease in the elimination of the dye. Two cases have been observed in which an acute nephritis was engrafted on an old kidney lesion. Both cases were in men in early adult life, both from their histories and physical examinations gave evidence of preëxisting chronic nephritis. In one, in whom on admission the urine contained 3 gm. per litre of albumin, casts and red blood cells, the 'phthalein was 11 per cent. After a week there was decided improvement in the urinary findings and the 'phthalein had risen to 22 per cent. In the other the 'phthalein was 17 per cent. on admission, 19 per cent. a few days later and finally increased to 21 per cent.

In the majority of acute infections the 'phthalein excretion has been but little below normal or even increased. In one typhoid patient with a trace of albumin and a few casts, the 'phthalein was 42 per cent.; in another it was 49 per cent. One case of typhoid fever that terminated fatally from profound toxemia, about one week before death showed a 'phthalein excretion of 85 per cent. Another. in whom undoubtedly a nephritis existed, as evidenced by a heavy cloud of albumin and casts, had a dye output of 39 per cent.

In lobar pneumonia the phenolsulphonephthalein elimination was found to be 48 per cent., 65 per cent., and even as high as 70 per cent, in spite of the presence of albumin and casts.

In acute articular rheumatism, 'phthaleins of 48 per cent. were met with, and in a fatal case of pulmonary tuberculosis with a persistent albuminuria but no casts, a 'phthalein of 42 per cent. occurred.

It would seem that in the main the 'phthalein excretion is an uncertain factor in many of the acute infections in which the functional activity of the kidneys undergoes such rapid variations and in which the permanent renal damage is usually slight. Given a case, however, in which the urinary output is falling and in which 'phthalein and other functional tests show persistently diminished excretion, and serious kidney failure should be anticipated and combated.

The test was of prognostic value in cases in which an irritant poison had been ingested. In a case of creolin poisoning in a young woman, the urine the day after ingesting the drug contained albumin and the 'phthalein elimination was only 15 per cent. in two hours. The urine promptly returned to normal and within a week the patient was discharged cured.

In one case of bichloride of mercury poisoning in a man, aged twenty-eight years, who swallowed one-half of a 1½-gr. bichloride tablet, the urine showed a trace of albumin and a few casts, but the 'phthalein was 49 per cent. and the patient recovered. In another case, however, in a woman, aged twenty-five, who had taken a large dose of bichloride, on admission the urine contained, a trace of albumin and the 'phthalein was 30.5 per cent. The following day there was a heavy cloud of albumin, casts, and red blood cells, and the 'phthalein output fell to 17 per cent. For the next week the urine contained 0.5 per cent. per litre of albumin, and casts, etc., and the 'phthalein was zero. For next three weeks this patient's urine was somewhat better, the quantity eliminated much increased, but the 'phthalein excretion continued at zero, or at best showed only in very faint traces. The unfavorable termination of the case bore out the grave prognosis which for some weeks had been indicated by the lack of 'phthalein excretion.

A case of iodine poisoning in a young girl also gave interesting 'phthalein findings. On admission the urine was normal and her 'phthalein 68 per cent. Ten days later the urine contained a trace of albumin, but no casts, and the dye output had fallen to 27 per cent. On discharge some days later, her urine was again normal and her 'phthalein was up to 47 per cent.

In decompensation from valvular or primary myocardial disease, varying grades of chronic passive congestion of the kidneys may result and the urinary picture may closely simulate a true nephritis. In such cases the 'phthalein test is of value as it enables one to obtain an approximate idea as to how much actual renal involvement exists. Our experience corresponds in the main with the statement of Rowntree[10] that in chronic passive congestion of the kidneys, due to failing compensation, the 'phthalein excretion is generally unexpectedly good. In a series of cases of chronic endocarditis it averaged between 43 per cent and 58 per cent. At times the excretion may be alarmingly low, purely from a congestion; as in one case in which it was only 7.5 per cent. in two hours, and the urine contained much albumin and many hyaline casts. At the end of ten days the urine was absolutely normal and the patient had subjectively recovered. In another case of passive congestion, the 'phthalein excretion at first was 32 per cent., but in two weeks had risen to 50 per cent.

From our own experience, as well as from the observations of others, certain conclusions in regard to the utility of the phenolsulphonephthalein test in medical cases seems warranted.

1. As a method of readily and accurately determining *total renal function*, the 'phthalein test is superior to any other single functional test, although it gives little or no information as to the kind of kidney lesion or the part of the kidneys most involved.

2. An accurate and reliable conception of the functional capacity of the kidneys cannot be arrived at by depending upon any single test. Conclusions of value can only be drawn when the condition of 'phthalein excretion is taken in conjunction with a complete clinical survey of the case and even then many conditions will arise about which exact knowledge can only be gained by supplementing the 'phthalein test by other excretory tests and a determination of "cumulative phenomena."

3. In our hands chronic interstitial nephritis has generally shown a persistently low 'phthalein output, although cases do exist in which a normal or almost normal 'phthalein excretion is found. Even an acute uremia may show a good 'phthalein elimination.

4. In cardiorenal cases, and cases of decompensation the 'phthalein reaction furnishes valuable data as to which factor is the dominant one, the heart or the kidneys.

5. In acute infections the 'phthalein output is usually fair.

6. In acute irritant poisoning cases, the 'phthalein test will give early evidence of serious kidney involvement.

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# LEUKEMIA: REPORT OF TWO CASES[1]

By CARL R. STEINKE, M.D.
FORMERLY SURGEON TO THE DISPENSARY OF THE HOSPITAL

Case I.—A. N. C., a white merchant, aged fifty-four years, married, had negative family and previous medical histories. The patient has one daughter, who is healthy. He was first seen February 23, 1913, complaining of pain in the cardiac and left hypochondriac regions and at times in the lower abdomen. His present trouble began one and one-half to two years previous and he had lost 20 pounds in the preceding six months.

*Physical Examination.* Skin and mucous membranes were pale; the thorax negative. The liver extended from the sixth rib to two finger breadths below the costal margin. The kidneys were not palpable. The spleen extended from the ninth rib to 1 inch above the iliac crest with sharp anterior border in the midclavicular line. The inguinal and axillary glands were palpable; no epitrochlear or postcervical glands could be felt. The musculature was flabby.

Urine: 35 ounces in twenty-four hours, was normal. Blood: red cells 3,725,000; hemoglobin 75 per cent.; leukocytes 179,480 with differential count of 70 per cent. polymorphonuclears, 20.3 per cent. myelocytes, 5 per cent. transitionals, 3.6 per cent. lymphocytes, 1 per cent. eosinophils, 1.6 nucleated reds to 100 leukocytes. No treatment was taken until September 27, 1913, when the liver dulness was about as before, while the spleen extended well into the left iliac fossa and to the left nipple line anteriorly.

*Treatment and Sequence.* Fowler's solution was given, running up to 12 minims three times a day, with no effect after several weeks' trial. Roentgen-ray treatment three times a week began January 28, 1914. On February 9 he noticed severe pain down the right thigh and leg, nocturnal in character and most marked between

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[1] Reprinted from the Journal of the American Medical Association, September 11, 1915, vol. lxv, pp. 954 and 955.

2 and 4 A.M., Nervous and rectal examinations revealed nothing abnormal. By March 30, after eighteen Roentgen-ray treatments, the spleen was considerably reduced in size. He discontinued treatment for two weeks and on return the spleen was larger than ever and his general condition much worse, thus showing the necessity of continuous treatment. Previously the various long bones as well as the spleen had been exposed to the Roentgen ray and Dr. Newcomet decided to expose only the spleen, focusing from various angles. On May 26, the blood count was: red cells 3,990,000; hemoglobin 70 per cent.; leukocytes 211,800; differential count, polymorphonuclears 49.38, myelocytes 20.5, basophils 19.62, large mononuclears 5.3, transitionals 2.1, eosinophils 1.8, lymphocytes 1.3. After forty-one Roentgen-ray treatments together with six weeks of benzol (benzene $C_6H_6$), the blood count improved as shown in the accompanying table. His temperature ranged between 100 and 103° F. and the pulse averaged about 80. From the middle of June the patient failed more rapidly. July 6 the urine showed a trace of albumin and many granular casts. The blood count was red cells 3,030,000; hemoglobin 44 per cent.; leukocytes 295,000; polymorphonuclears 28.5, large mononuclears 63, myelocytes 7.5, transitionals 0.5, basophils 0.5; nucleated reds present. Death occurred July 12, 1914, of respiratory failure. The great increase of large mononuclears toward the end of life has been observed by others.

Necropsy at the Presbyterian Hospital gave the characteristic features of splenomyelogenous leukemia, but there was no local cause found for the pain down the right limb.

BLOOD COUNT AND TREATMENT IN CASE I.

| Date. | Hb% | R. B. C. | W. B. C. | Myelocytes, % | Treatment. |
|---|---|---|---|---|---|
| Feb. 23, 13 | 75 | 3,725,000 | 179,480 | 20.3 | None. |
| Sept. 27, 13 | 73 | ........ | 204,880 | 30.0 | Fowler's solution. |
| Jan. 28, 14 | .. | ........ | 240,000 | .... | Roentgen ray started. |
| Mar. 30, 14 | 80 | ........ | 164,000 | .... | Benzol given five days. |
| May 5, 14 | .. | ........ | ...... | .... | Benzol started again. |
| May 26, 14 | 70 | 3,990,000 | 211,800 | 20.5 | |
| June 12, 14 | .. | ........ | ...... | .... | Benzol stopped. |
| July 6, 14 | 44 | 3,030,000 | 295,000 | 7.5 | |

Case II is reported by courtesy of Dr. M. H. Fussell from his service at the Episcopal Hospital. Acute lymphatic leukemia: F. U., white, aged 11, had negative family and previous medical histories. No pneumonic history was obtainable. He complained of cough, shortness of breath and a "swollen" chest. He began feeling ill two weeks before admission.

*Physical Examination.* This showed a well-developed, well-nourished boy with swollen face, enlarged tonsils, coated tongue, enlarged cervical nodes. Thorax expansion was good on the left but much diminished on the right, where the enlargement, which did not pit on pressure, appeared. The veins over the anterior chest were numerous and dilated (telangiectasis). The right chest had signs of fluid and 2 c.c. of this pus were aspirated which gave a negative bacterial smear and culture, while the leukocytes (mostly lymphocytes) were too numerous to count. On the left side the breath sounds were exaggerated, vocal fremitus increased, but there were no râles or friction sounds. The heart was negative. The liver was not enlarged and the spleen not palpable. The extremities were negative. The leukocyte count was 12,160, polymorphonuclears 70 per cent., lymphocytes 22 per cent., mononuclears 5 per cent., transitionals 2 per cent., eosinophils 1 per cent. A roentgenogram of the chest was negative for empyema. Urine: amber, specific gravity, 1.025, acid, contained no sugar, albumin or sediment. Ephithelial cells and urates were present, and a few leukocytes, but no red blood cells or casts. The temperature ranged from 98 to 100° F. By August 20 the liver dulness extended from the fifth rib to 1½ inches below the umbilicus in the midaxillary line and the spleen was easily palpable. The abdomen was rigid but not tender. The inguinal nodes were enlarging. Expectoration was bloody and gums bleeding. Fowler's solution, 5 minims three times a day, was given and Roentgen-ray treatments were started.

August 26. The blood count was: reds 4,980,000, hemoglobin 82 per cent., leukocytes 67,500.

August 29. Leukocytes 256,800, lymphocytes 93 per cent., polymorphonuclears 6 per cent., eosinophils 1 per cent. His condition rapidly grew worse so that by September 1, the following special features of his condition were noted: He had an anxious expression, his face was puffy, he had marked dyspnea, petechiae on inner side of feet and bluish discoloration of soles, spine bowed anteriorly, discrete enlargement of submaxillary, epitrochlear, and axillary nodes on both sides, inguinal nodes palpable. The right chest gave signs of fluid, with the apex beat in midaxillary line. There was a large mass in the upper abdomen, probably the spleen or liver, as the two could not be differentiated. One and a half pints of bloody serous fluid were aspirated from the right chest and it was still flowing when violent coughing necessitated the withdrawal of the needle. Laboratory report on the fluid gave 10,000 red blood cells and 160,000 white blood cells with a differential count of 98 per cent. lymphocytes and 2 per cent. polymorphonuclears. A smear showed a few diplococci, but the culture was negative and no tubercle bacilli were in the fluid (guinea-pig test made). The blood-count was: red cells 3,000,000, leukocytes 215,000, small lymphocytes 90 per cent., and polymorphonuclears 10 per cent. The gums oozed freely. The patient died suddenly at 6 A.M., September 3, 1914. Unfortunately no necropsy was obtained.

The case conforms to R. C. Cabot's[1] description of acute lymphatic leukemia. In the cases reported by Drs. Fussell, Jopson and Taylor,[2] a number showed an increase in the percentage as well as the actual number of small lymphocytes.

*Treatment.* The Roentgen-ray and benzol (7 minims in seven minims of olive oil in capsule three times a day) treatment in the first case (splenomyelogenous leukemia) had a very effective result, and had treatment started early and continued faithfully, the condition could at least have been controlled. In the second case (acute lymphatic leukemia) with such a rapid course, no known treatment would have been of avail.

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[1] In Osler, Modern Medicine, 1908, iv, 671, 673.

[2] Philadelphia Med. Jour., January 7, 1899.

# BLOOD TRANSFUSION—THE MODIFIED BREWER'S TUBE—REPORT OF CASES

By J. WALKER MOORE, M.D.
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Blood transfusion has become an important therapeutic entity in recent years. While it was a recognized procedure as early as 1824, it fell into disuse through the influence of Senn and other writers of his day by their advocacy of what was thought to be a more efficient and much simpler procedure—namely, normal saline transfusion into the vein. Thus, it remained for Crile, in 1906, to revive an operation which had become all but extinct.

The cannula method introduced by Crile threw aside the terror of embolism and infection to which the old operation subjected the patient, and opened wide a field hitherto unknown to medical science. Thus, recent investigators in blood dyscrasias, animal surgery, etc., have equipped us with numerous simple and efficient methods for blood transfusion. As a result, transfusion is no longer looked upon as a procedure preliminary to summoning an undertaker. It has a definite rank in treatment of certain medical conditions; it holds a place unparalleled in the preparation of exhausted cases where operation is imperative; and its value in post-operative cases cannot be overestimated.

The methods that are more commonly used in blood transfusion may be divided into direct and indirect.

Direct Method. The direct method signifies the direct communication between the vessels of the donor and the recipient, either artery to vein or vein to vein.

Artery-to-vein transfusion is best carried out by Crile's cannula, or its modifications, and the modified tubes of Brewer and Fauntleroy.

In vein-to-vein transfusion, Crile's cannula, modified glass tubes of Brewer and Fauntleroy, and Price's forceps cannula (*Lancet-Clinic*, January 25, 1913) give excellent results.

THE INDIRECT OR SYRINGE METHOD. David and Curtis have devised a very ingenious procedure for blood transfusion by the syringe method (*Jour. Am. Med. Assn.*, March 7, 1914). The apparatus consists of a large glass bulb, a capacity of 400 c.c., with two cannula tips. To assist the flow of blood a two-way valve syringe is attached to the bulb. The bulb is coated with melted paraffin. The two cannula tips of the bulb are attached, one to the vein of the donor and the other to the vein of the recipient. The artery clamp is released from the vein of the donor and the bulb allowed to fill with the aid of negative pressure produced by the syringe. The recipient's vein is released and the donor's vein clamped, and the blood is introduced into the former at any desired rate of flow.

Coley, Edwards and Vaughan advised a method which consists of a 20 to 50 c.c. glass syringe and two large bore needles with trocars. The needles with the trocars are inserted into the veins of the donor and recipient. The trocar is removed from the needle of the donor and the blood drawn into a 50 c.c. syringe containing 10 c.c. of a freshly prepared normal saline solution, the latter being used to delay coagulation. The syringe is disconnected and the trocar placed into the needle. The trocar is now removed from the vein of the recipient, and the syringe connected with the needle and emptied. This procedure is carried out until a sufficient amount of blood is transfused.

The Kimpton-Brown tube is considered by Mason (*Surg., Gynec. and Obst.*, 1915, xx, 737) to be the simplest method so far devised for transfusion. The special instruments required are the Kimpton-Brown tube and a cautery bulb. The one great advantage claimed for this method is that donor and recipient are not brought together as in the anastomosis methods.

The glass-tube method which we have used with success employs a modification of Brewer's transfusion tube.

The tube (Fig. 5), three-sixteenths of an inch in diameter, is bent in such a way that the two arms, pointing in opposite directions, make an angle of 45 degrees with the main axis of the tube, the axis measuring 5 cm. and the arms 2 cm. each, the entire length of the tube being 9 cm. The ends of the tube are slanting and burnished, the arterial end being smaller than the venous end, with a slight though distinct bulging 2 mm. behind the opening. The slanting facilitates the entrance of the tube into the vessel and the bulging provides a shoulder which prevents the slipping of the vessel after tying. To further facilitate the entrance of the cannula into the radial artery, the arterial end of the cannula is beveled and burnished. It is seldom necessary to have the venous end beveled.

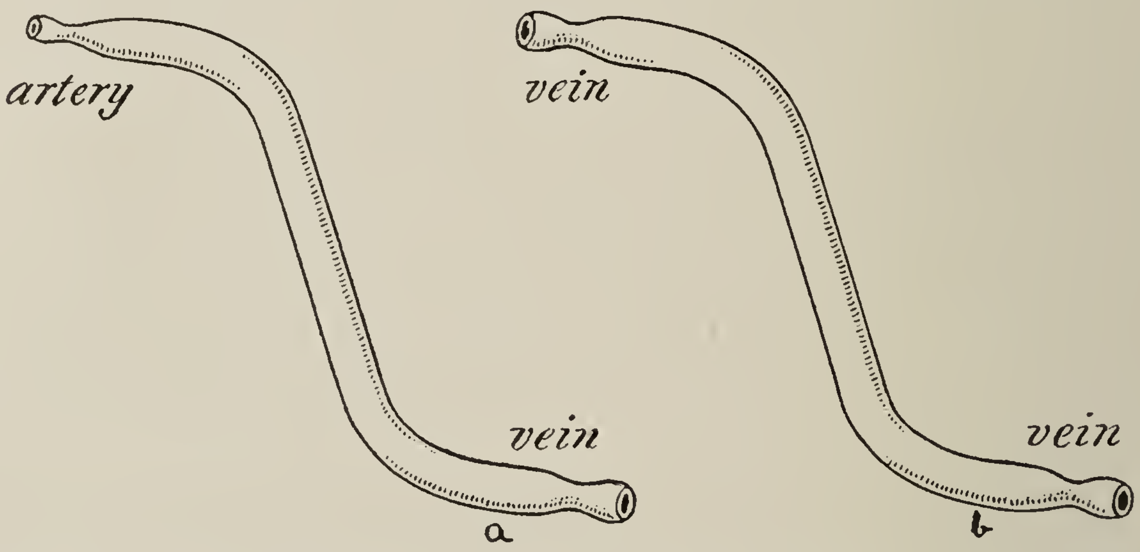

Fig. 5.—Actual size and shape of transfusion tubes: *a*, artery-to-vein method; *b*, vein-to-vein method.

Tubes of any desired length can be easily made with Bunsen's burner. For adult transfusion the length shown in Fig. 5 will suffice in the majority of cases. However, stock collections of varying sizes and lengths are recommended.

The shape of the tube obviates the necessity of long incision and free dissection of the vessel, which undoubtedly reduces the chance of infection.

Technique of the Operation. The dry sterile tubes are dipped in sterile melted paraffin or Vincent's mixture (paraffin 2 parts, vaselin 2 parts, stearin 1 part) and shaken out to remove the excess of paraffin or the Vincent's mixture. If a drop clings near the end it can easily be removed by touching it gently with a piece of sterile gauze. The tubes are now placed flat in a dry sterile vessel and the paraffin allowed to cool.

When the *artery-to-vein* method is desired, the donor and recipient are placed side by side in opposite directions. The arms are sterilized and a tourniquet applied around the upper part of the recipient's arm just tight enough to distend the vein. The area of incision is anesthetized by injecting eucaine, 2 per cent. solution, over the line of radial artery in donor, and cephalic vein in the recipient. With a small incision the artery and vein are exposed, care being taken not to dissect them out of their bed, as their being anchored facilitates the entrance of the tubes and prevents angulation of the vein. Two linen or chromic gut ties are threaded on an aneurysm needle and passed beneath the vessels, one above the other below the portion to be nicked, and the distal ligatures on both artery and vein are tied. A pair of blunt-pointed, curved scissors are pushed beneath the radial artery near the upper angle of the wound and a rubber-covered clamp applied to the artery at this point. The artery is cut by scissors transversely half way through with a slant from below upwards. With mosquito forceps the small flap of the artery is retracted, the tube inserted and tied in place. The arms of the patients are approximated in hand to axilla direction, with the forearm of the donor held in midposition and the recipient's forearm in full supination (Fig. 6). A nick is now made in the vein and this is held open with mosquito forceps, the tourniquet is removed, the artery clamp released and blood allowed to pass through the tube, which is then quickly placed within the lumen of the vein and secured by a ligature. Pulsating waves can be seen passing through the tube and felt in the vein near the tube.

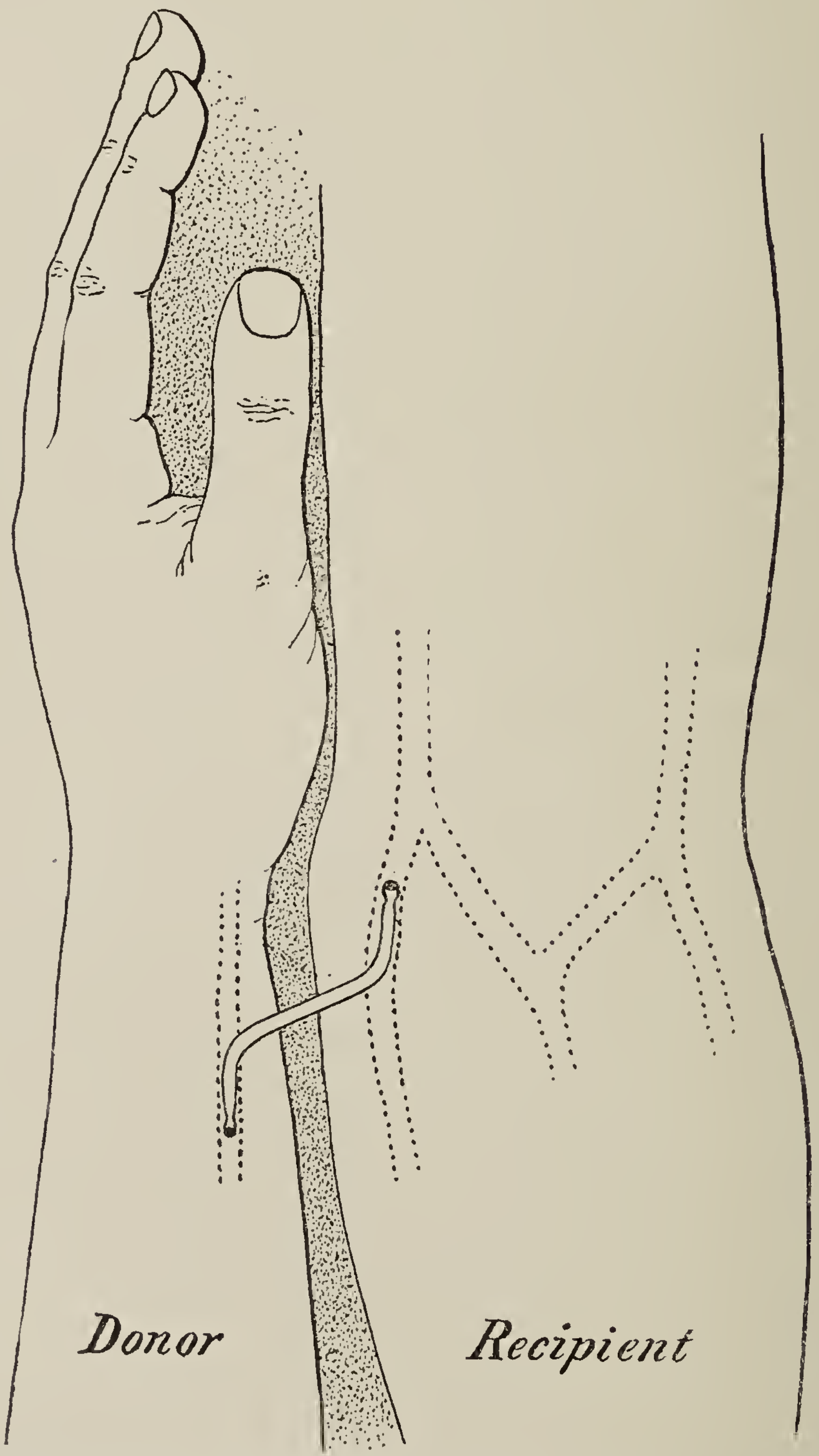

Fig. 6.—Diagram showing tube in use for artery-to-vein transfusion.

*Vein-to-vein* transfusion requires the same technique as is carried out in artery-to-vein transfusion. In this procedure the donor and recipient are placed side by side in the same direction, with shoulders together. The veins in front of the elbows are used. A tourniquet is applied above the elbow of the donor tight enough to produce marked venous hyperemia in the forearm. The tourniquet must not cause stoppage of the radial pulse. This procedure is simple and angulation or twisting of the veins should not occur, as the veins are not dissected out of their beds; as a result, clotting is reduced to a minimum. One can tell that the blood is passing by the improvement in color, in pulse, and by the fulness of the vein near the tube.

The artery-to-vein method is the more desirable of the two procedures. In the first place, one is sure that the blood is passing by seeing the wave in the glass tube and feeling the pulse in the vein. Secondly, the procedure is much more rapid than the vein-to-vein method, and the desired effects are reached within a few minutes, and this, I must say, is a big item in urgent cases. I believe that in the third case of transfusion herein reported, the patient would have survived if the artery-to-vein method had been ccarried out rather than the vein-to-vein procedure. Thirdly, angulation or twisting is less likely to occur.

The amount to be transfused in either method will depend upon the reaction of both the donor and the recipient. The flow should be stopped as soon as the donor shows symptoms of anemia, as respiratory embarrassment, rapid pulse, etc. In the event the recipient is brought into a satisfactory condition before the untoward symptoms of the donor are reached, the flow should be stopped.

A more elaborate method has been devised by Libman and Ottenberg (*Jour. Am. Med. Assn.*, March 7, 1914) for estimating the amount of blood the donor may spare. This method is of great value and should be used whenever possible, but it is not absolutely necessary.

Case I.—Wm. K., male, aged fourteen years, Episcopal Hospital. The patient was anemic, pale, waxy in color, and nothing more than skin and bones from the interminable suppurative process that had followed an injury to the lower third of the femur, several months previously. On August 14, 1914, two secondary hemorrhages occurred from sinuses along the course of the femoral vessels and it was thought advisable to amputate the thigh at the junction of the middle and upper thirds. Red blood cells, 1,540,000; hemoglobin, 20. In his condition amputation meant death, so as a last resort transfusion was decided upon. The mother agreed to give her blood. The artery-to-vein method, using the modified Brewer's tube, was carried out. During the process of the transfusion, performed by Drs. Jones and Moore, the lips and ears of the patient became pink and then red, pulse fuller and stronger. During the transfusion the patient was given nitrous oxide gas and the thigh amputated by Dr. A. P. C. Ashhurst. He made a remarkable recovery.

Case II.—R. H., male, aged twenty-seven years. Admitted into Dr. Frazier's service in the Episcopal Hospital, September 1, 1914, with a history of having vomited a quart of bright red blood the day before, and a pint on the day of admission.

The patient was anemic, pale and exceedingly restless, with a profuse clammy sweat over the entire body. Lungs were clear. Respirations rapid and shallow. Pulse 127, of low tension and volume. Aside from exaggerated peristalsis, the abdomen was entirely negative. Red blood cells, 2,840,000; hemoglobin, 42. Color index, 75.

The day after admission he vomited a quart or more of bright red blood, together with a few dark red clots. Morphia was given in sufficient quantities to stop peristalsis. Patient complained of intense thirst. Extremities were cold and the body temperature below normal. Pulse was soft, small, rapid and at times could not be detected.

A donor is selected and artery-to-vein transfusion carried out in the usual manner by Drs. Jones and Moore. Within a short time the color returned into the blanched lips and the luster to the eyes. At the end of the procedure, which was twelve minutes in length, the pulse was full and bounding, color returned to the face and extremities and the patient was in excellent spirits.

Just before transfusion, R. B. C., 1,820,000; Hb., 30; color index, 83. Just after transfusion, R. B. C., 3,110, 000; Hb., 60; color index, 96. Six hours later, R. B. C., 3,350,000; Hb., 61; color index, 94. Twenty-four hours later, R. B. C., 3,275,000; Hb., 65; color index, 94. Forty-eight hours later, R. B. C., 3,333,000; Hb., 60; color index, 90.

*Of Donor.* Before transfusion, R. B. C., 5,300,000; Hb., 97; color index, 96. After transfusion, R. B. C., 4,410,000; Hb., 80; color index, 91. Six hours later, R. B. C., 4,600,000; Hb., 85.

Patient picked up rapidly in general condition, so much so that one week later (Hb. 62 per cent.) the abdomen was opened. An ulcer was found in the anterior surface of the stomach one and one-half inches from the pylorus, one inch below the lesser curvature, directly in the course of a large vein. With linen suture the ulcer is invaginated and a posterior gastro-enterostomy performed by Dr. Ashhurst.

Case III.—Case No. III is Case No. II one week after the abdominal operation. The patient did nicely until one week after the abdominal section, when, with only a few minutes warning, he vomited a large pus basin full of bright red blood. This was the first time he had vomited since the transfusion two weeks previous. He continued to vomit blood the entire day, even though Morphia, horse serum, etc., was pushed to the limit.

A donor for blood transfusion was sought, during which time the patient was rapidly sinking. At each respiration the nostrils opened widely and the accessory muscles of respiration were put into action. The pulse was no longer perceptible at the wrist. At this point the donor, who had given his blood two weeks ago, offered to give more blood provided a vein instead of the radial artery be used. The two were rushed into the operating room and a vein-to-vein transfusion performed by Drs. Drane and Moore. The patient was totally unconscious when the blood began to flow. The transfusion was stopped at the end of fifteen minutes, as the patient showed no evidence of improvement. He succumbed thirty minutes later.

At autopsy numerous acute ulcerations in the mucous membrane of the stomach in the region of the pylorus, on its posterior surface, were found. The ulcers varied from the size of a pin head to a pea.

CASE IV.—H. B., male, aged twenty-four years. Admitted into Dr. Frazier's service in the Episcopal Hospital October 22, 1914. The patient received a gunshot wound of outer side of right thigh at the juncture of the upper and middle thirds. Forty minutes later he was brought into the hospital by a patrol. On admission the patient was profoundly shocked. His entire body was pale and cold, pupils dilated, reacting slowly to light. The respirations were shallow and rapid, The pulse weak and rapid. There was a steady oozing from the wound and the surrounding clothing saturated with blood. Red blood cells, 1,600,000; hemoglobin, 20.

The *x*-ray showed the femur to be shattered about the juncture of middle and upper thirds. A tourniquet was applied above the wound and the classical treatment of shock carried out. The patient's condition rapidly grew worse. The pulse became imperceptible at the wrist. Blood transfusion was advised. His sister agreed to give the blood. The artery-to-vein method was carried out in the usual manner by Drs. Drane and Moore. The transfusion was discontinued after seven and one-half minutes of flow, as there was no evidence of improvement. A pint of normal saline was given into the exposed vein. The patient never reacted, and died two hours later.

I wish to express my great indebtedness to Dr. Charles H. Frazier, in whose service at the Episcopal Hospital the patients were treated, for the privilege of reporting the cases.

# KERATOSIS PALMARIS ET PLANTARIS, WITH REPORT OF A CASE[1]

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This rather rare condition is very much like callositas, differing in the fact that the hardening and thickening arise spontaneously without friction or pressure.

It is confined to the palms and soles usually and is symmetric. The distal ends of the nails may be elevated by a collection of thickened epidermis. In most cases there is marked hyperidrosis of both hands and feet. At the border of the diseased part there is usually a red area, which is not due to inflammation. As a rule, the entire sole and palm is covered by a tough callus, which may be from one-eighth to one-quarter of an inch in thickness, the arch of the foot being the only part to escape. In some cases, as in the patient shown to-night, the lesions may be linear and cause contraction of the fingers.

The only subjective symptoms are interference with free motion and pain when walking, owing to the great thickening.

Etiology. Nothing is known beyond the fact that it is congenital and sometimes hereditary. Several cases have been reported in the same family and in different generations.

Pathology. It is a hardening and thickening of the corneous layer of the skin.

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[1] Read at a meeting of the Episcopal Hospital Clinical Society, September 21, 1914.

Diagnosis. The age of the patient, the symmetric character of the lesions, the hyperidrosis and the lack of inflammation will distinguish it from ordinary callositas. Arsenical keratosis might be confused with this disease, but the history of the case should render the diagnosis easy.

Treatment. To date nothing has been discovered that will make a permanent cure, although many remedies have been suggested. A strong salicylic acid application seems to do more in the way of keeping the growth down than anything else. *X*-ray seems to have failed in the majority of cases.

Case.—S. M., aged fifteen years. Born in Philadelphia.

*Family History.* Father and mother, nine brothers and two sisters are living and well. One sister died in infancy. No history of keratosis in the family.

*Personal History.* Negative.

*Present Condition.* On the palmar surface of the right hand there is a tough thickened area, extending from the base of the thumb to the last phalanx of the third and fourth fingers. There is less involvement of the fifth and second fingers. The left hand shows the same process but to a lesser degree. Both feet are involved and are thickened from the base of the toes to the heel.

Since admission to the hospital the patient has been anesthetized and the growth scarified and fibrolysin injected into the sound and diseased skin of the right hand and the affected skin of the left hand has been excised by Dr. Ashhurst, who covered in the raw surfaces thus exposed by plastic operations (September 11, 1914).

What the result will be is problematical, but at present there seems to be some improvement in the right hand; the left hand has healed and is becoming useful.

# ZOSTER ARSENICALIS, WITH A REPORT OF THREE CASES

By RICHARD S. HOOKER, M.D.
PHYSICIAN TO THE DISPENSARY OF THE HOSPITAL

The relationship between zoster and the use of arsenic was first noted by J. Hutchinson in 1868. Ten years later the observations were corroborated by Dyce, Duckworth and others, but it was looked upon by many as a mere coincidence. Neilson, in 1890, however, conclusively proved that arsenic can produce zoster. He states that in 390 cases of psoriasis treated with arsenic 10 developed zoster, while among 220 cases of psoriasis otherwise treated zoster was not seen.

As late as 1907, Zeisler, in a paper before the Sixth International Dermatological Congress, says: "Nor is there perfect unanimity among observers concerning the causal relationship between the administration or ingestion of the drug and the subsequent cutaneous reaction. While some entertain no doubt as to the direct connection, a few still consider zoster arsenicalis merely as a coincidence."

My idea in reporting the following three cases is: (1) to draw attention to one of the rarer manifestations of arsenic upon the skin, the existence of which, in my experience, is little known except to dermatologists, and one which, like many other rare diseases, or complications, may cease to be so unusual when more generally recognized; and (2) to add to the number of reported cases and so give additional weight to the evidence showing a relationship between zoster and arsenic.

Case I.—James H., aged fifty-four years, born in England. Used to be a machinist, but for the last four years has worked as a fleecer. His family history is negative. Has always been considered strong and healthy. Had typhoid fever ten years ago. Denies venereal disease. Has never used alcohol to excess. Smokes moderately.

Four weeks ago he first noticed that he had lumps under each arm, also in groins, about the size of a peanut. Sometimes they are very hard and large, and at other times they seem smaller and softer. They have gradually increased to their present size.

Patient is a well-developed and well-nourished man of fifty-four years. There is marked enlargement of the lymphatic glands of the neck and axilla and groin. The axillary glands are largest, being about the size of an orange. General examination, excepting for glandular enlargements, was negative.

Admitted May 12, 1914, and Fowler's solution, 5 minims, t. i. d., prescribed. Blood examination showed red blood cells, 4,140,000; hemoglobin, 82 per cent.; white blood cells, 20,480; polymorphonuclears, 31 per cent.; lymphocytes, 45 per cent.; mononuclears, 23 per cent.

May 23. A small lymph node was removed from the back for examination, and it gave a typical picture of lymphosarcoma.

May 28. Began an *x*-ray treatment three times weekly to the glandular areas.

July 5. There was no apparent change in his condition.

July 7. He developed a very marked supra-orbital herpes zoster of the left side, several patches becoming gangrenous. The left eye was much inflamed, and lymphatic glands of the neck much enlarged during zoster attack.

He had been in the hospital for fifty-six days, during which time he had been steadily taking Fowler's solution, 5 minims, t. i. d.

Case II.—Carrie C., schoolgirl, aged twelve years, white, born in United States.

Family history is negative, excepting that one sister died of chorea, complicated by diphtheria, and one sister died from spasms.

Has had measles. Operated on a few months ago for appendicitis. Has had stomach trouble since eighteen months old. This is much better since operation for appendicitis. Been in school since her eighth year.

Her present trouble began about three weeks ago, with pain over the entire left side. Two days later the left arm began to twitch. A few days later the left leg began to twitch also. These movements have continued to get worse.

Examination shows a well-nourished and well-developed girl of twelve years of age, with very active choreic movements of the left arm and leg. Excepting for slight roughening of the first sound at the apex, which is transmitted toward the sternum and anterior axillary line, there is nothing abnormal.

On admission to the hospital April 14, 1914, she was placed in bed, given a purge, and Fowler's solution, 5 drops, t. i. d., was prescribed. She improved slowly until April 24, when she had an attack of tonsillitis, and movements became worse again. This subsided in two days and the condition began to improve rapidly. On May 1 heart sounds were normal and movements almost stopped. On May 6, twenty-two days after admission, an eruption typical of herpes zoster developed on the left chest. This cleared up in a few days and the patient was discharged cured on May 23, 1914.

Case III.—James C., aged twelve years, schoolboy, white, born in United States.

Family history negative. Mother declares the only other illness he has ever had was measles. During the past three months mother states that she has noticed the boy becoming more nervous and that he has complained of pain in the various joints at times.

On admission on August 10, 1915, he was found to be a fairly robust-looking boy. Choreic movements were general and extremely active. Tonsils cryptic. Marked vitiligo of the face and scalp. There is a systolic murmur heard at the apex and transmitted to the axilla.

He was given sodium salicylate, 10 grains, every four hours. This was stopped on August 17, and Fowler's solution, 5 drops, three times daily, given.

His improvement was very slow but uneventful, and he was discharged on September 21, 1915, cured. He had been taking Fowler's solution, 5 drops, t. i. d., for thirty-five days.

Eleven days later, October 2, he developed a marked attack of herpes zoster, right buttock especially in the anal sulcus, also right half of scrotum and penis; there were no lesions across the median raphe. He was again admitted to the hospital on October 4, 1915.

This case illustrates one of the reasons, I think, why this complication is not more frequently seen. The patient often does not develop zoster until two or three weeks after being discharged from the hospital, and is usually treated with home remedies or by another physician, who does not connect the zoster with the previous sickness and medication.

# A TREATMENT FOR OLD CONTRACTED CICATRICES[1]

By EMORY G. ALEXANDER, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

There is scarcely any condition in surgery that so tests the patience, ingenuity and skill of the operator as the treatment of old cicatricial contractures. Especially is this true if the cicatrices are hard, dense and of long duration. It is not my object to review what has been done in the past in the field of plastic surgery, as the method of procedure in each case must be determined on the local findings. In one case the division of contracting bands may suffice, in another the sliding, twisting or transplantation of flaps, to take the place of the excised cicatrices may be required. In still other cases, especially of the extremities, the deformity may be so great as to require amputation.

Are bad cicatricial contractures following burns as frequent as they were a generation ago? Our present-day textbooks on surgery would lead us to believe that they are not, as little attention is given to this very important subject. Possibly we have improved in the treatment of burns, certainly, antiseptic dressings and early skin grafting has reduced to a large degree the late bad effects.

It is often difficult and sometimes impossible in cases of cicatricial contractures to get healthy flaps, and if one be so fortunate as to succeed, the result is always in doubt, as the flap may slough on account of a deficient blood supply.

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[1] Read before the Philadelphia Academy of Surgery, March 2, 1914. Reprinted from Annals of Surgery, October, 1914.

In old, dense cicatrices, the result of deep burns, plastic operations are not without risk, as important structures, such as nerves and bloodvessels may be caught in the scar tissue and their anatomical situation so disturbed as to render injury almost unavoidable.

The case I wish to present tonight was not treated by the usual method of plastic surgery. In this case a plastic operation has been done at the wrist and axilla with much improvement; at the elbow, where an old firm cicatricial contracture existed, a good result has been obtained. This result has been obtained, not by the usual method of dissecting out the cicatrices and the lateral approximation of healthy skin, nor by the sliding, twisting or transplantation of flaps, but by operative, medicinal, and mechanical means.

Case.—E. F., aged seventeen years, white, was admitted to the Episcopal Hospital on May 16, 1913, with the following history:

At the age of fourteen years he received a severe burn of the left side of the body, arm, forearm and hand. The patient was eighteen months recovering from the burn. He was treated for ten and one-half months as an inmate in a local hospital in the town in which he lived, and for seven months after being discharged from the hospital, in their dispensary.

A physical examination of the patient on admission to the Episcopal Hospital showed the heart, lungs and other organs to be healthy. The left wrist was in extreme flexion, the joint ankylosed, and the overlying skin and cellular tissue consisted largely of scar tissue and contracting bands. The fingers could be freely moved. The thumb was partially ankylosed. The left elbow was markedly flexed and held by contracted bands of hard cicatrices. The left arm was held close to the chest wall by a large web of contracted scar tissue. The shoulder, chest and abdominal wall showed contracted cicatricial tissue.

On June 12, 1913, a plastic operation was performed at the wrist, as his hand was so markedly flexed as to render it useless. At this operation an arthrectomy (first row of carpals removed) and a plastic operation were performed. The result was quite gratifying, as shown in Fig. 7. An arthroplasty on the wrist is the next operation we intend to perform. One month after the above operation the patient was discharged from the hospital in the condition as shown in Fig. 7.

On November 14, 1913, he was again admitted to the Episcopal Hospital, and on November 17, 1913, was operated upon and a plastic operation of the flap, sliding type, was performed on his axilla, the flap being gotten from the pectoral region. On his arm and forearm a different procedure was tried, many incisions were made along the contracting bands. These incisions extended into the healthy skin laterally and were of varying depth, some being quite superficial and others being deep enough to penetrate the cicatrices. After the wounds had ceased to ooze, fibrolysin, a compound of thiosinamine and sodium salicylate, was next rubbed thoroughly into each incision and also injected hypodermatically into the fibrous tissue. The wounds were covered with rubber tissue and a dry dressing applied. A straight splint, padded mould-shape to take up the concavity caused by the flexed elbow, was applied. The bandage holding the splint was firmly applied, so as to extend the forearm as much as possible.

For two days following the operation the patient complained of pain at the elbow. This was undoubtedly due to the extension made on the forearm.

In one week's time quite an improvement was noticed. The forearm could be extended fully 50 per cent. further than it could before the operation and the cicatrices were soft and pliable.

On December 4, 1913, the operation as described above for the administration of fibrolysin was repeated. After this operation the patient experienced nothing like the discomfort which followed the first. In ten days' time the arm was as straight as you see it now (Fig. 8). At each operation the contents of four ampoules of fibrolysin were rubbed into the incisions and injected into the cicatrices.

The after-treatment has consisted in the occasional injection hypodermatically of the fibrolysin into the site of the old cicatrices. Passive motion and massage have also been given. Three and one-half weeks following the second operation the splint was removed, but as the forearm showed a tendency toward flexion it was reapplied.

It is as yet too early to say what the ultimate outcome will be, as the tendency to contraction after the splint was removed has made me a little skeptical. No reactions were noted from the use of the drug.

Richardson, *Annals of Surgery*, December, 1911, in an article entitled "Studies on Peritoneal Adhesions," discusses

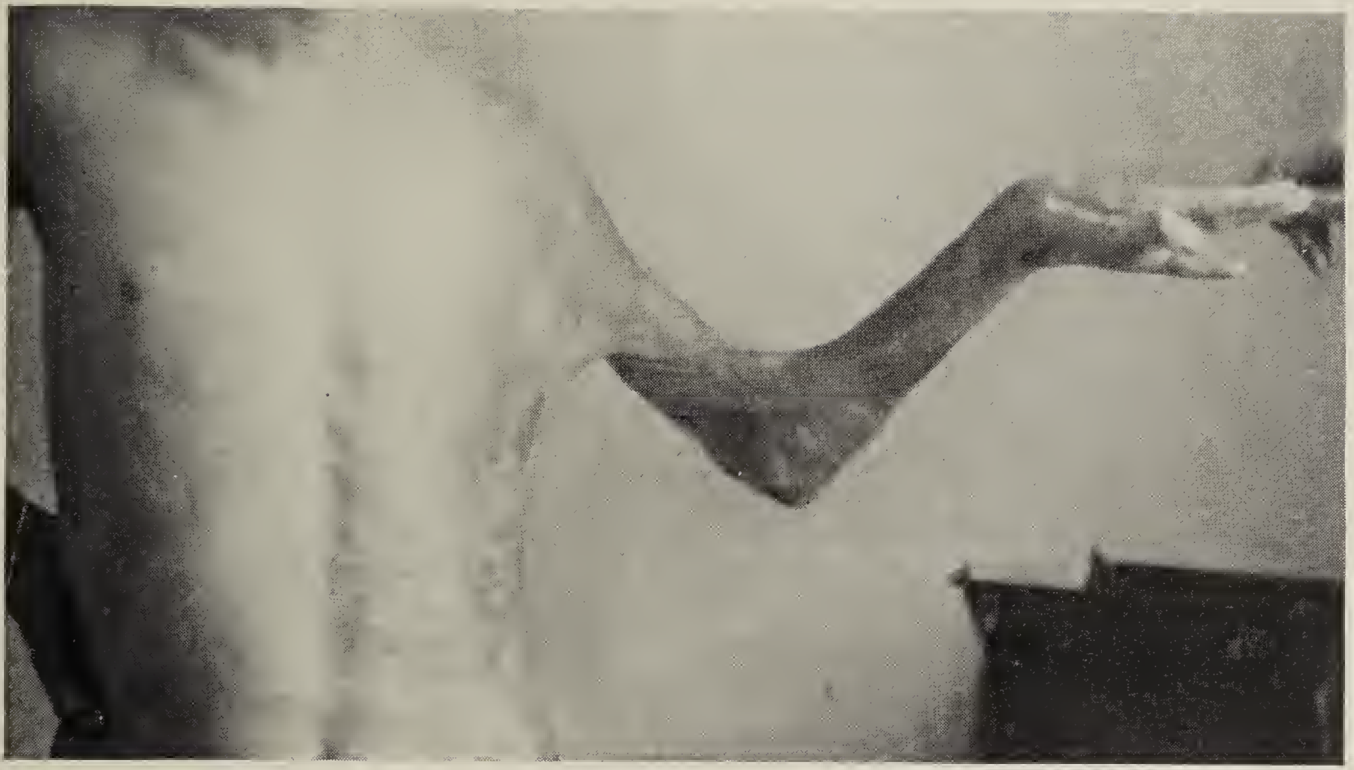

FIG. 7.—A plastic operation has been done at the wrist. Axilla and elbow show dense, contracted cicatrices.

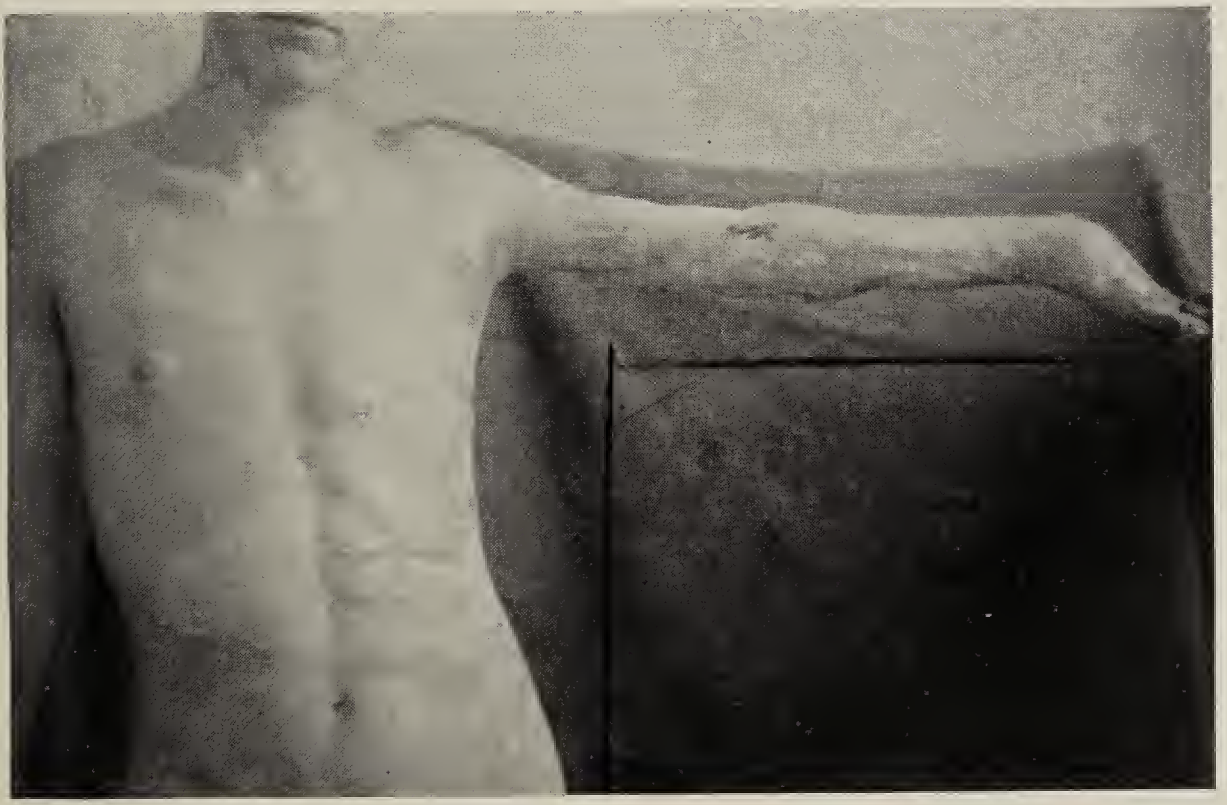

FIG. 8.—End-result.

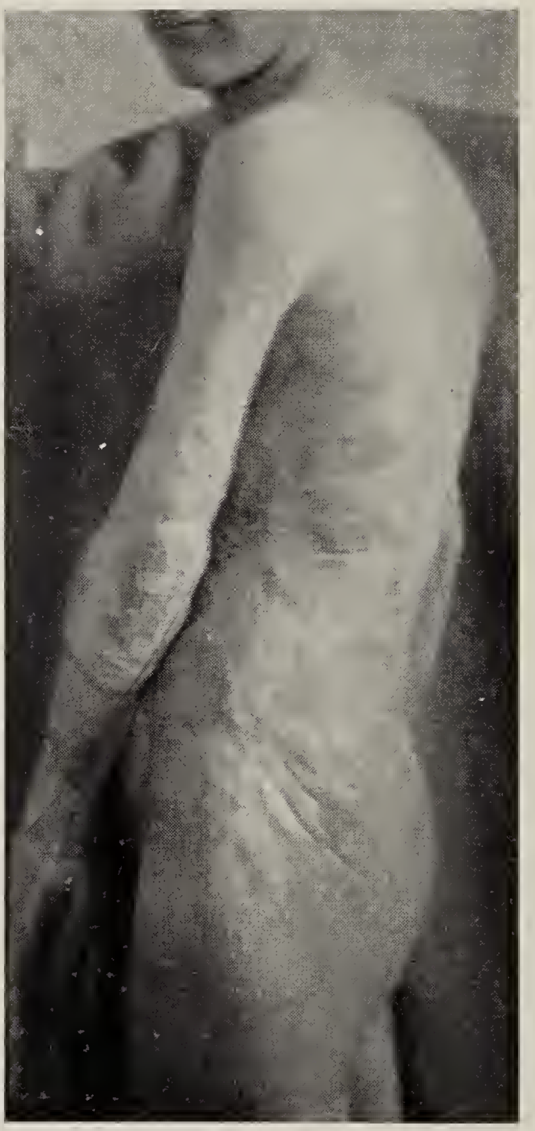

FIG. 9.—Back view.

the results obtained by specific drugs in the treatment of adhesions and cicatrices. He says, "Sidorenko has just reported the results of a clinical, experimental, and histological study of fibrolysin on cicatricial tissue. From a critical study of the results of other workers, and from his own results, he concludes that it does not exert any therapeutic effect upon cicatricial tissue."

A. H. Tubby, *British Medical Journal*, November 8, 1913, described a method of treatment of Dupuytren's contracture by flap dissection, excision of the affected fascia and the use of fibrolysin. While not following Mr. Tubby's technic, it was from his article that I got the idea of using fibrolysin in the way as described in this case.

Mr. Tubby, in speaking of his operative results in the treatment of Dupuytren's contracture, says, that in every case they were "infinitely superior" to those in which the thiosinamine (fibrolysin) was not used.

In the case just presented it is difficult to attribute the success to any one thing, as three distinct measures, operative, medicinal and mechanical were used. That the cicatrices were rendered soft and pliable lends weight to the drug.

# RUPTURE OF THE BICEPS FLEXOR CUBITI, WITH A REPORT OF EIGHT CASES[1]

By EMORY G. ALEXANDER, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

Rupture of the biceps muscle or its tendon is a rather rare accident. It may be of interest to the Academy, therefore, to present eight of these cases, especially as five were operated upon and the exact location of the rupture was determined. Literature reviewed on the subject cites only 74 cases, 5 of which were operated upon.

Case I.—M. C., aged twenty-six years, white, male, laborer. Admitted to the Episcopal Hospital January 9, 1914.

*History.* On the day of admission, while at work cleaning out an endless chain elevator shaft, the elevator suddenly started and one of the buckets caught his right arm, crushing it against the side of the shaft. He was held in this position for forty-five minutes before being liberated. The shock following the accident was so great that it was necessary to remove him to the hospital in the ambulance. On admission to the hospital the patient was in a state of severe shock. He was unable to raise the right arm or flex the forearm.

*Examination.* Showed a contused wound of the right chest wall and right arm. No bones were broken. The right arm anteriorly from the shoulder to the elbow was markedly swollen and ecchymosed. On deep palpation over the upper part of the biceps, where the swelling was greatest, a distinct furrow could be felt. This furrow was about the width of the index finger. The width of the furrow could be increased by extending the forearm. On account of the great swelling of the arm no bulging appeared at any point over the biceps on extending or flexing the forearm. A diagnosis of rupture of the belly of the biceps was made and an operation advised. On account of the bruised condition of the tissues the operation was postponed for several days.

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[1] Read before the Philadelphia Academy of Surgery, January 4, 1915. Reprinted from Annals of Surgery, May, 1915.

On January 11, 1914, under ether anesthesia, the patient was operated upon and a rupture of the belly of the short head and a partial rupture of the belly of the long head of the biceps were found. The ends of the muscles were properly approximated and sutured with twenty-day chromic catgut. The arm was dressed with the forearm in flexion, so as to relieve any strain on the sutured muscle. Primary union followed. Firm union took place between the ruptured ends. This could readily be determined by grasping the biceps over the seat of rupture and flexing and extending the forearm. On account of the marked atrophy of the humeral group of muscles which followed the accident, the return of power in the patient's arm was very slow, and massage and electrical treatment were given. At the time of his discharge from the hospital the biceps still showed marked atrophy, but the patient had good use of the arm and forearm. The usefulness of the arm has steadily improved and the patient is now able to follow his usual occupation.

Case II.—F. L., aged fifty three years, white, male, cloth finisher. Admitted to the Episcopal Hospital on January 16, 1914.

*History.* In 1910, while putting a large belt on a fly-wheel, he lost his balance and fell, striking his right shoulder and arm against a machine. When he struck the machine he felt something give way in his arm, experienced a sharp pain at this point, and heard "a sound like the report of a revolver." Immediately his arm became swollen, "black and blue," and a "lump" appeared at the lower part of the arm over the belly of the biceps. For a short time following the accident he was unable to use the arm. He consulted a physician for the injury, who prescribed a liniment. No fixation apparatus was applied. The ecchymosis and swelling disappeared in one or two weeks, but the "lump" remained. He continued working at his trade but his arm was so weak that he finally gave up his position and sought an easier one. Since the accident he has been able to do only light work, as his forearm under muscular effort will often give way. The weakened condition of the arm and forearm has persisted, although he has been treated with electricity, massage, etc. He has never had rheumatism, gout, typhoid fever nor lues.

*Examination.* His right arm, when either the forearm was flexed or extended, showed a marked bulging of the belly of the biceps. The bulging was greatest when the forearm was forcibly flexed to a right angle and extension strongly resisted. The entire biceps muscle was soft and flabby. Above the belly of the biceps, which terminated very abruptly, it was impossible to feel the tendon of the long head. There was no difference in the power of flexion of the forearm, whether it was supinated or pronated. A diagnosis of rupture of the tendon of the long head of the biceps was made. As four years had elapsed since the accident it was with some hesitancy that an operation was advised, but as the patient was practically incapacitated it was hoped that an operation might better his condition.

On January 19, 1914, under ether anesthesia, he was operated upon. An old rupture of the tendon of the long head at its junction with the belly was found and fibrous union had taken place between the tendon and the muscle. This cicatricial tissue, about one and one-half inches, was excised. The end of the tendon was split for about three-quarters of an inch and the muscle brought in between the two ends of the split tendon and sutured with twenty-day chromic gut. The arm and forearm were dressed as in Case I. Primary union followed.

The case progressed most favorably and the patient now states that he has been much benefited by the operation. A slight bulging still persists at the site of the old rupture.

CASE III.—P. McC., aged sixty-one years, white, male, punch helper. Admitted to the Episcopal Hospital on March 21, 1914.

*History.* The patient came to the Medical Dispensary of the hospital on account of pain (rheumatism) in his right shoulder and weakness of his arm. In examining him the physicians found that he had, besides a chronic articular rheumatism, a rupture of the biceps muscle. Although the bulging was very great the patient had never noticed it. The patient stated that about six weeks before he came to the dispensary for treatment, while at work in a punch shed, he tripped and fell, striking his right shoulder. The injury caused him some pain and was followed by some stiffness of the shoulder. Since the accident he has noticed that his arm and forearm are weaker and that the weakness is increasing.

*Examination.* Symptoms similar to Case II, the only difference was that in this case the bulging was greater and the muscle more flabby. Hüter's sign was not present.

On March 26, 1914, the patient was operated upon and a rupture

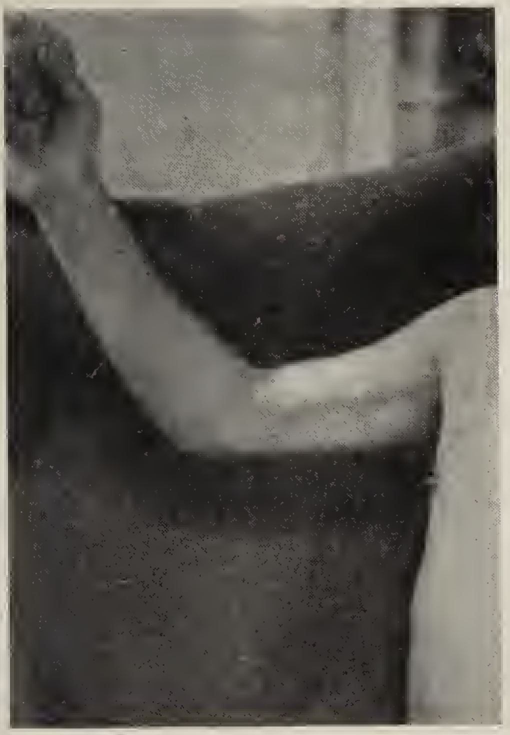

Fig. 10.—Case I. Five weeks after operation.

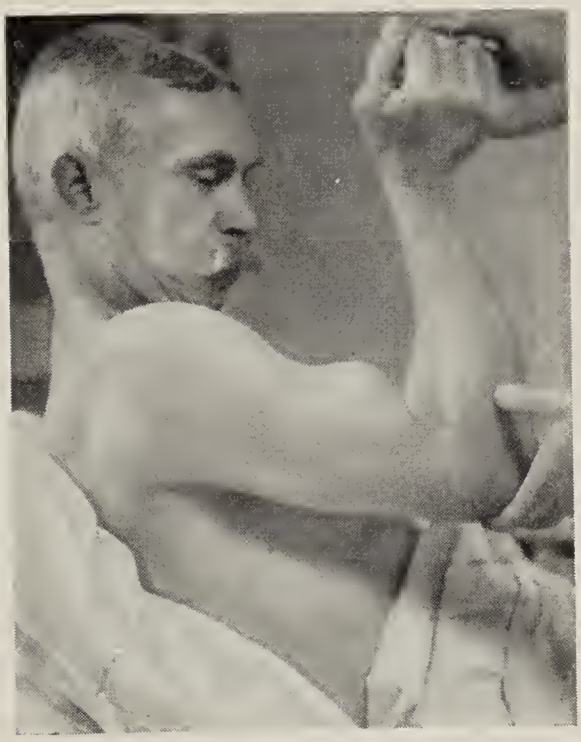

Fig. 11.—Case II. Before operation.

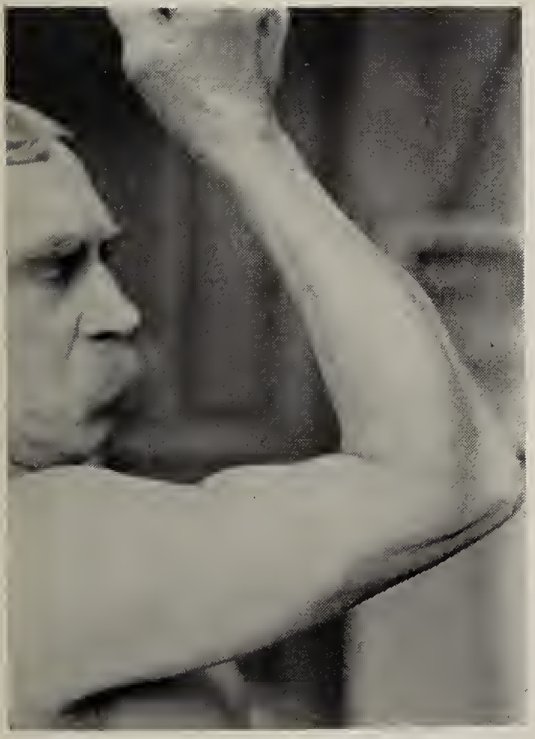

Fig. 12.—Case II. End-result.

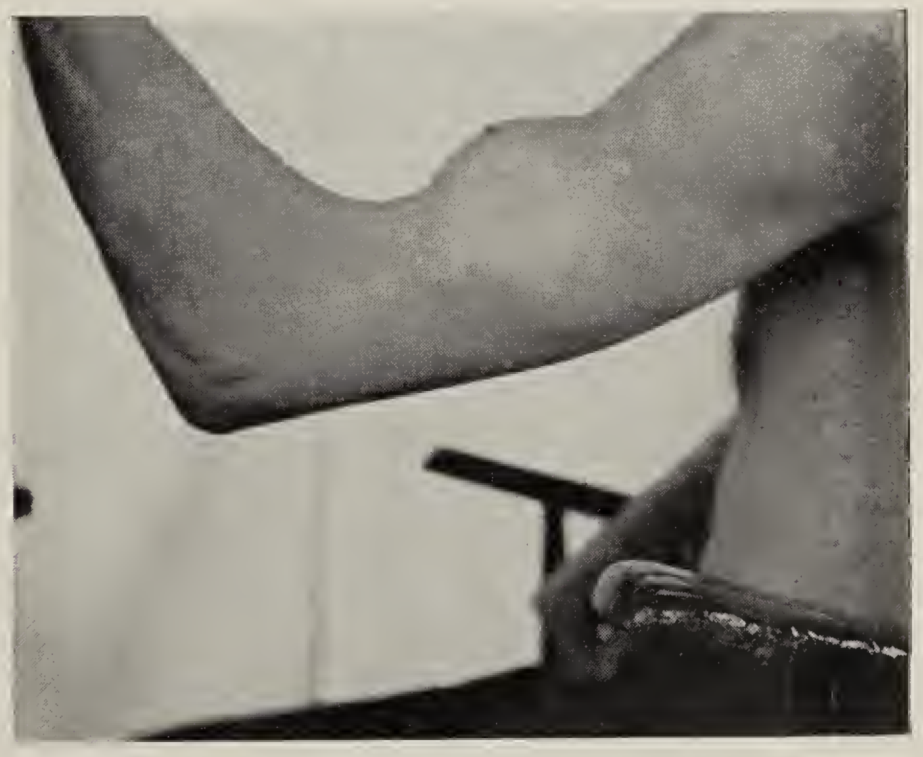

Fig. 13.—Case V. Before operation.

of the tendon of the long head at its junction with the belly found. I was unable to find the tendon of the long head although I searched for it up to the bicipital groove. I did find a fibrous sheath, which I took to be the sheath of the tendon; there was, however, no tendon within it. As the sheath was not strong enough to suture the muscle to, the stump of the belly of the long head was sutured to the tendon of the short head.

The patient's arm was treated as in Case II. Primary union followed. Anatomically, the patient does not appear to be very much benefited, as he still has quite a bulging of the muscle.

Functionally, he has been greatly improved and now says he is sure he is able to return to his former work, which he had had to give up on account of the weakness of his arm.

This patient, I feel sure, ruptured his biceps muscle much longer ago than six weeks before the operation. The interesting point is that although he had quite a bulging of the muscle he had never suspected any trouble.

Case IV.—G. C., aged fifty-three years, white, male, machinist. Admitted to the Episcopal Hospital April 17, 1914.

*History.* Six months before admission, while working on a lace curtain machine, he attempted to stop the wheel by catching it as it revolved. He succeeded in stopping the wheel, but it gave his shoulder quite a "wrench" and he felt something "give way" in his arm and heard "something snap." On flexing the arm he noticed a bulging over the biceps. Although he noticed that his arm was much weaker the accident did not completely disable him. The weakness was progressive and for this reason he came to the hospital. There was no history of gout, rheumatism, typhoid fever or lues.

*Examination.* Symptoms similar to Cases II and III. Hüter's sign was present. (Hüter calls attention to the fact that flexion of the forearm in pronation when the biceps is tense is more forcible than when the forearm is supinated and the biceps is relaxed.) On April 20, 1914, under ether anesthesia, the patient was operated upon and a rupture of the tendon of the long head at its junction with the belly found. The tendon was readily found, but it appeared more as a small fibrous cord than as a tendon. On account of the weakened condition of the tendon, I did not think it advisable to suture it to the muscle, but instead, sutured the belly of the long head to the tendon of the short head. The case was treated as the preceding. Primary union followed. The patient has a very satisfactory result. There is very little bulging of the muscle on forced flexion of the forearm and the arm and forearm are much stronger than before the operation.

Case V.—W. S., aged forty-one years, white, male, laborer. Admitted to the Episcopal Hospital on December 9, 1914.

*History.* On the morning of admission to the hospital, while lifting a bag of coffee, he was suddenly seized with pain and heard something snap in his left arm. He said it felt as if "somebody had hit him with a club." As he was unable to use his right arm after the accident, he came immediately to the hospital. There was no history of typhoid fever, gout or lues, but an indefinite history of a mild attack of rheumatism in the left shoulder.

Symptoms were those of a rupture of the long head of the biceps. No hematoma or other signs of recent injury were present. Hüter's sign was not present.

The patient refused to be operated upon and left the hospital the same day that he applied for treatment.

This patient was operated upon one week after the accident by Dr. G. G. Davis at the University Hospital. The operation disclosed the tendon of the long head relaxed. A rupture had taken place at some point between its origin and the bicipital groove. The tendon was not pulled out of the groove, but the slack portion was taken up and the tendon sutured to the tendon of the short head.

Case VI.—R. McF., aged sixty years, white, male, fish dealer. Admitted to the Episcopal Hospital on December 15, 1914.

*History.* Three months before admission, while attempting to lift a barrel of fish, he was suddenly seized with a severe pain in his left arm. He dropped the barrel and rested the arm for a few minutes until the pain subsided. The night following the injury he suffered greatly with pain in his arm, forearm and fingers. Since the accident, on account of the weakness of his arm, he has been unable to do heavy work. He says that his left arm frequently "goes to sleep." No history of typhoid fever, rheumatism, gout or lues, but is a heavy whisky drinker.

*Examination.* Symptoms similar to Cases II to V inclusive. Hüter's sign not present. The muscle showed quite a bulging and was very flabby. Blood-pressure 180 systolic. No symptoms of nerve involvement.

*Diagnosis.* Rupture of the long head of the biceps, probably at the junction of the tendon and muscle. As the patient was in very poor physical condition no operation was advised.

Besides the 6 cases reported above, two other cases have been treated this year in the Medical Dispensary of the Episcopal Hospital.

Case VII.—Wm. M., aged eighty-four years, white, male, insurance agent. This case came to the Dispensary on account of a traumatic arthritis of the left knee. In the examination of the patient it was discovered that he had a rupture of the long head of the biceps muscle of the left arm. The probable seat of rupture was at the junction of the muscle and tendon. The Dispensary notes state: "No symptoms at time of rupture or afterward; no history of rheumatism, typhoid fever or lues."

Case VIII.—Wm. L., aged fifty years, white, carpenter. The patient was admitted to the Dispensary for treatment for chronic endocarditis and myocarditis. He was treated six years ago in the Episcopal Hospital for a severe attack of acute articular rheumatism. The lesion in this case was of the long head of the biceps of the left arm. The rupture, as in Case VII, was probably at the transition point between tendon and muscle. This case, as the preceding, had "no symptoms at time of rupture or afterward."

Unfortunately, in the above 2 cases, no note was made as to whether the patients were right- or left-handed.

Loos[1] reports 66 cases of rupture of the biceps, 44 of which were of the muscle, and 18 of the tendon. Four of these were his own.

Petit,[1] according to Loos, collected 83 cases. In this series the point of rupture was as follows: Muscle 21; belly of the long head 9; tendon of the long head 43; at junction of belly of the long head and tendon 7; the tendon of insertion 3. I failed to find an article on the subject by Petit, so was unable to verify the statistics as given by Loos.

Wiesmann[2] reviews the literature on this subject and reports a case of rupture of the tendon of insertion caused by the lifting of a table. He operated upon the case and obtained a successful result.

Davis[3] reports 4 cases, as follows: Rupture of the tendon of the long head 3; rupture of the tendon of insertion 1.

Castret[4] reports a case of rupture of the belly of the biceps due to slight muscular contraction. The case was treated by electricity.

Hollos[5] reports "two cases of biceps rupture by paralysis." I have been unable to find his article.

Keen[6] reports a case of rupture of the tendon of the long head and also one of DaCosta's, of a tearing off of the long head from the margin of the glenoid cavity. Both of these cases were successfully operated upon.

Gerster[7] reports a case of laceration of the long head of the biceps due to a fall down stairs. The case was successfully treated with a sling.

In 66 of the cases reported, in which the histories were given in sufficient detail to locate the lesion, 16 occurred in the tendon, 44 in the muscle, and 6 at the junction of the tendon and the muscle. As only 5 of the cases reported were operated upon the exact site of the rupture in the others could not be ascertained.

Davis[3] believes that true rupture of the tendon is rare and "when a tendon does rupture it is very likely to have been diseased." In Case IV of this series it appeared as if the tendon was either diseased or malformed.

From the cases reported it appears that rupture may take place through the belly of the long or short head, through the belly of the muscle proper, at the transition point of the belly and tendon, through the tendon of the long head or the tendon of insertion, and even as in DaCosta's case, at the origin of the tendon of the long head.

Of the cases reported, 4 have been the result of muscular effort alone. Only 2 cases are reported in which the accident occurred in women. Only 2 cases are reported of rupture of the belly of the short head; one of these was due to direct injury, a threshing machine accident, in which the belly of the short head and the tendon of the long head were both injured, an accident very similar to Case I of this series. The other case occurred in an attempt to reduce a dislocated shoulder.

The causes of the rupture may be either direct force, muscular contraction, or possibly an indirect force, such as a fall on the shoulder, that would throw a great strain on the long tendon; also disease or malformation might be an underlying cause.

Since the great majority of the cases occur among working men, and since the right biceps is the one usually affected, it seems that muscular contraction is the most potent etiological factor.

In Cases II, III and IV of this series the rupture occurred at the transitional point between tendon and muscle. As tendon is stronger than muscle it is natural to believe that the rupture occurred on the muscle side of the tendon and that the underlying cause might possibly have been a myositis. This cause is suggested in Case III, where the entire muscle was markedly relaxed and very flabby, although only the tendon and belly of the long head were implicated in the accident. The cases reported show that rupture is most apt to occur after forty years of age. As this is the period that degenerative tissue changes are most apt to make their appearance it is very probable that disease of muscle or tendon plays a very important causative part in the rupture.

That rupture of the tendon or belly of the long head is more frequent than at any other site, I believe to be due to the fact fact that the tendon, by its position in reinforcing the shoulder-joint, is subjected to all strains that may be thrown upon the head of the humerus. Cases II and III may have occurred in this manner.

The symptoms at the time of injury may be trivial, the patient noticing nothing wrong, or they may be quite severe and immediately incapacitate. Swelling and ecchymosis do not always accompany the rupture.

The physical findings will differ somewhat, depending on the site of the rupture. If the rupture is through the belly of the muscle a furrow can be felt between the two ends, this can be widened by extending the forearm. In rupture of the tendon of insertion, flexion and supination of the forearm may be interfered with and the belly of the muscle would be drawn up nearer the shoulder.

In rupture of the long head, whether it be a tearing away at its point of origin, in the continuity of the tendon, at the transition point between tendon and muscle or through the belly of the long head, the symptoms are almost identical. A bulging of the biceps at a point nearer the elbow than normal, a flabby condition of part or all of the muscle, an abrupt termination of the bulging above and an inability to feel the tendon above that point, all point to rupture of the muscle or tendon. In rupture of the tendon or muscle of the long head the short head stands out prominently, and to its outer side, where the long head and tendon should be, a sulcus running upward and slightly inward is found.

Hüter's symptom was present in only one of this series. It was not tried for in Case I.

The treatment in the great majority of cases has been either none at all, or bandage, sling, electricity, etc. No doubt many cases have been operated upon. I have only been able to find, however, 5 cases treated in this manner.

Von Hochstetter[8] reports a case of a man, aged forty-six years, who ruptured the tendon of the long head at its junction with the belly. This case was operated upon two months after the injury. The tendon was sutured to the belly with silk. Result successful.

Bazy[9] reports a man, aged forty years, who ruptured the tendon of the long head at its point of origin. He operated upon this case, resected the tendon, and sutured the stump to the tendon of the short head. Result successful.

Keen[6] reports a case of a man, aged fifty-four years, who ruptured the tendon of the long head. The tendon was sutured with twenty-day chromic gut. Result successful.

DaCosta,[6] reported by Keen, operated upon a man, aged fifty-two years, who in lifting a bucket tore the tendon of the long head away from its point of origin. In this case the upper portion of the tendon was resected and the stump was attached to the short head by splitting the latter and suturing. Result successful.

Wiesmann[2] reports a case of rupture of the tendon of insertion, caused by lifting a table. The case was successfully operated upon.

Conservative methods of treatment have given satisfactory results in a few isolated cases only. Since successful results have been gained by operation in cited cases, it is evident that the operative treatment is to be recommended.

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# THE PREVENTION AND TREATMENT OF THE DISABILITIES FOLLOWING FRACTURES OF THE LIMBS[1]

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DURING the last few years I have had under my care a great many patients suffering from disabilities due to fractures of the limbs. This has impressed me with the frequency and seriousness of such disabilities; and as the first step toward their prevention is a knowledge of their origin, I venture to bring before you this subject, which I believe is of prime importance. In fact, I cannot but believe that, taking fractures of the limbs in general, the results of treatment are less satisfactory than they used to be. And I think this is so not alone because we have advanced our standards of what shall be considered good results, but also because our treatment is less efficient than in former years. This state of affairs, perhaps, may be attributed to the comparative lack of interest in, and neglect of proper instruction of students concerning the treatment of fractures during the decade, let us say, from 1900 to 1910. This was a period during which the interest of the surgical side of the profession was concentrated largely upon abdominal surgery; it was a vast and fascinating subject, which had just begun to reach its stage of mature development, and in society meetings, clinics, and the class room swept everything before it.

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[1] Read by invitation at a meeting of the Brooklyn Surgical Society, February, 4, 1915. Reprinted from the American Journal of Surgery March and April, 1915.

During this wave of enthusiasm interest in dry bones lagged, and it was not until within the last four or five years that bone surgery, and especially the subject of fractures, began to be restored to its former state of importance. The consequence is that most students of medicine who began their practical work during the abdominal decade knew little or nothing, and cared less, about the pathology or treatment of fractures.

It is unfortunately true that in the present state of civilization in this country, as indeed everywhere else in the world, it is still necessary for the general practitioner to have under his care every year a number of cases of fracture; he simply cannot avoid it. No doubt it would be much better for the patients if all cases of fracture could be referred to surgeons taking special interest in such work; and I think it is not to be disputed that whenever possible it is the duty of the family physician to send such cases to such a surgeon, and not to attempt to treat them himself. This course of action, however, is possible only in populous communities; and the grave responsibilities which must be assumed by the country practitioner, who must indeed be not a "jack" but a "master" of all trades in the profession, render his lot most unenviable.

But as it is impossible to know in advance which students of medicine will be able in after life to avoid having fractures under their care, it is necessary to insist that all shall receive adequate instruction in this, as in all other fundamental branches of medical science and art. I believe the tendency toward specialization is to be condemned at least until after the hospital years; and during these hospital years it is the duty both of those in charge of out-patients and of those in charge of the wards to train the Resident Physicians most carefully in the diagnosis, prognosis and treatment of fractures. Too much reliance, in particular, should not be placed on the *x*-ray, in diagnosis and prognosis. That young doctor whom you see depending entirely upon a skiagraph in a doubtful case, may be a few months hence hundreds of miles from the nearest $x$-ray outfit and may have to depend upon his $x$-ray fingers in treating his patients. It is mere folly for anyone to state, as Lane has done, that it is a pure waste of time to teach students the classical signs for the diagnosis of fracture, since the only thing needed is a good skiagraph.

So, too, in regard to the operative treatment of recent fractures. It is generally recognized that this method cannot be applied successfully even by the average general surgeon; much less by the occasional operator or country practitioner. Indeed, the operative treatment of fractures deserves almost to be considered a specialty; it is difficult manual labor, requires extraordinary care in the preservation of asepsis, and necessitates a special armamentarium possessed by few general hospitals and by no general practitioners. No one who does much work in fractures fails to see every year one or more lamentable results due to operative treatment of recent fractures by the inexpert.

It is on all these accounts doubly incumbent on those who are responsible for the theoretical and practical instruction of medical students to so train them in what may be truly called conservative methods of treatment, that they will be able without resort to operation to cure their patients with the minimum of disability. For a number of years I have been urging this very thing upon the profession: that non-operative treatment not only possesses the inestimable advantages of carrying out the sound surgical maxim "*primum non nocere*" but also may be employed by any sensible practitioner with a reasonable degree of success; and when employed with intelligence, thoroughness, and enthusiasm, in fractures almost of any type, and in long series of consecutive cases, will give as good as if not better results than will be secured by operation, done by the average surgeon, and without unnecessary risk to the patient.

Before discussing in detail the prevention and treatment of disabilities following fractures of the limbs, I may lay down certain theses, which will be developed in the sequel:

1. Lesions of the soft parts always accompany fractures of bone, but too often are ignored in treatment, and are a very frequent cause of disability.

2. In compound fractures lesions of the soft parts always should take precedence over those of the bones.

3. Active movements and functional use of the limb are the most important factors in overcoming disabilities due to lesions of the soft parts. Massage, especially *effleurage* and *pétrissage*, is of much value in relieving edema, overcoming stiffness in neighboring joints, etc.; but passive movements usually are useless and often are harmful.

4. When the fracture occurs in the shaft of a long bone, it is not always possible (without open operation) to secure anatomical reduction of the fragments, and in most cases such reduction is unnecessary.

5. When the fracture occurs near a joint, it is indispensable to secure anatomical reduction and this should be obtained by open operation if necessary.

6. If operation is necessary this fact should be determined within a week or ten days of the accident, and operation should be performed before vicious union occurs or bone atrophy develops.

7. If operative treatment is required for old diaphyseal fractures, it is because of non-union or because of concurrent lesions of the soft parts, and scarcely ever because of malunion of the bones.

8. Malunion rarely requires operative relief except when occurring in the immediate neighborhood of joints.

9. Non-union is not always an indication for operation, and operation should be delayed until it is certain that cure will not take place without it.

## I. DISABILITY FROM LESIONS OF THE SOFT PARTS

In every fracture there is contusion, ecchymosis, extravasation, and often muscular laceration, accompanying the osseous injury; and the disability which follows the fracture, in a large proportion of cases, is due to *neglect of the soft parts*, while attention has been concentrated upon the bony deformity.

Yet the surgeon must not neglect the osseous lesion in his devotion to the care of the soft parts. There is a happy medium between the school of Lucas-Championnière, which ignores the bones and devotes all its attention to the soft parts by massage and by mobilization of the neighboring joints, and the modern operative school which teaches that so long as the ends of the broken bone are brought into absolute anatomical reposition the soft parts may be safely left to take care of themselves. The former school has the merit of recognizing that the bones are not sticks or stones or bars of iron, but that they are living tissue, and if given even a slight chance will rectify their own deformities in many instances and so produce no lasting disability; while the operative school is too prone to ignore primary lesions of the soft parts, and to blame all subsequent disability not on these (the real cause) but on insignificant osseous distortions appreciable only on skiagraphic examination.

It is not my purpose now to describe in detail the methods to be employed in proper care of the soft parts, but I may state as a general rule that for the first week or ten days the condition of the soft parts should receive paramount consideration, especially when the fracture is not close to a joint. When the fracture is close to a joint there is no doubt that the easiest means of relieving the swelling of the soft parts is to secure anatomical reduction of the fragments as soon as possible; and this no doubt is true also though in less degree in the case of fractures in the shafts of long bones. But in the latter instances it is very much more difficult to secure accurate reduction without open operation than when the fracture is close to a joint, and malunion of a diaphyseal fracture *per se* is very rarely a cause of disability. Hence I believe that in diaphyseal fractures attempts at reduction sometimes may be postponed for a few days until the swelling begins to subside. Up until the end of the first week no insuperable obstacle to reduction will develop, unless it was present also from the very moment of the accident, as for example, entanglement of the fragments in the soft parts.

Proper care of the soft parts requires that the dressing be removed two or three times weekly (less often as the case advances) to permit cleansing of the skin; and as union progresses to allow of very gentle passive movement of the neighboring joints. Such passive movements should never cause pain, and as already indicated should be substituted by active movements so soon as union of the bones permits.

One of the most frequent causes of disability due to neglect of the soft parts is edema of the limb below the seat of injury. Of course such edema is not always avoidable in the lower extremity, but in the upper limb it is nearly invariably an evidence of neglect. A common and perfectly unjustifiable error is neglect to bandage the hand and forearm in cases of fracture of the humerus, or even neglect to bandage below the wrist in cases of fracture in this situation. In treating a Colles's fracture of the radius, for instance, it is no unusual fault for the dresser to leave the hand and fingers totally unsupported by the bandage, no turns of which pass below the thumb; the result is that the hand and fingers become very edematous, and stiffness, pain and marked disability persist in the fingers long after the bone has united. Similarly, in fractures of the humerus, the dresser will make all secure from the shoulder to the elbow, but will leave the hand and forearm bare; they then become edematous, and long after the shoulder is healed, the patient must be treated for stiffness in the wrist and fingers. Only last summer I had to keep such a patient under treatment for more than three months after the shoulder fracture ceased to trouble her, because she was totally incapacitated by the condition of her hand; even more incapacitated, indeed, than if her hand had been amputated for those three months, since she suffered much discomfort from its presence.

In the lower extremity, as I have said, swelling of the foot cannot always be prevented during convalescence, owing to its constantly dependent position; but I am sure much less disability would result on this score if the soft parts of the lower extremity received, as is their due, two or three times weekly, proper "dressing" as in the case of fractures in the upper extremity, instead of being imprisoned for weeks at a time, as is usually the case, in circular gypsum cases.

Perhaps the most conspicuous example of lesions of the soft parts overshadowing those of the bones, is to be found in cases of Volkmann's contracture. Though this seriously disabling condition occasionally has developed in cases where no dressings at all have been employed, it is the result, in the vast majority of instances of constriction from too tightly applied splints or bandages, particularly when these are not removed for some days or weeks at a time. It should be an invariable rule for the surgeon to see the patient the next day after the fracture, and personally to examine the condition of the finger tips and make certain that there is no interference with the circulation, and that the dressing is comfortable. An uncomfortable dressing always is inefficient even if not positively harmful.

## 2. COMPOUND FRACTURES

There is no class of fractures in which care of the soft parts assumes such importance as in compound fractures. Here the bones are quite a secondary consideration; and even in cases where immediate formal operation is required it is usually inadvisable to apply plate or other form of internal fixation until asepsis can be assured by definitive healing of the soft parts.

I cite below several illustrative cases, in all of which the state of the soft parts made it proper for the first dressing to assume the character of a major operation.

Case I.—*Compound comminuted fracture of radius and ulna, rupture of ulnar artery and nerve, etc.*

Fred B., aged twenty-eight years, was admitted to Dr. Frazier's service in the Episcopal Hospital, December 12, 1909. His left forearm had been crushed between two freight cars while he was engaged in coupling them. All four ends of the broken radius and ulna projected through a jagged wound of the soft parts, the skin being torn loose all around the circumference of the limb except for 4 cm. over the extensor surface of the radius, and the flexor muscles being extensively ruptured. There was loss of sensation in the distribution of the ulnar nerve below the injury. I operated about eight hours after injury. The ulnar artery was crushed and its ends widely separated; both ends were ligated. The ulnar nerve was crushed for a distance of 4 cm., only the fibrous sheath remaining intact. By dissecting the proximal end even to the elbow, and the distal end to the wrist, and by flexing the wrist, it was impossible to make the ends of the ulnar nerve meet by 1.25 cm. Therefore a flap was turned up from the distal segment of the nerve, about 3 cm. in length, and sutured to the central end, without tension. The ends of the radius were drilled, and united by a suture of chromic gut No. 3. The ulna was found to be broken in two places, the intervening fragment being composed of three splinters. Silver wire was wrapped around these splinters, and they were sutured to the lower fragment of the ulna by chromic gut, passed through drill holes. A rubber drainage tube was passed across the flexor surface of the bones, and the ruptured muscles were carefully repaired. The limb was kept vertical, and alcohol douches were given as often as the dressings became dry.

The attempts to secure fixation of the bones were not very successful, as is evident from skiagraphs made soon after operation; these give one the impression that the bones might have been struck by lightning. There was prolonged but never very active suppuration and sloughing, and the three splinters composing the middle fragment of the ulna were soon discharged as sequestra. Bony union occurred in the radius, but only fibrous union in the ulna. About a year after the accident, and when the soft parts had been healed for more than eight months, a sequestrum worked loose from the radius. Though an excellent result was obtained eventually, I believe even the little that was done to the bones at the time of operation tended to delay repair.

Compare, for instance, the much more rapid recovery in the following even severer case.

CASE II.—*Crush of forearm, double compound fracture of the humerus.*

Fred H., aged thirty-five years, was admitted to my service in the Episcopal Hospital May 27, 1913. His right arm had been caught in machinery, and he had been knocked up against the ceiling and then flung across the room. Here also all four ends of the broken radius and ulna projected from a gaping wound in the flexor surface of the forearm; there was also a double fracture of the humerus of the same side, and one of the fragments of the humerus was projecting through the skin, on the inner side of the arm, below the axilla.

*Operation* was immediate. Fortunately no arteries or nerves were injured; only the basilic vein, transfixed by the humeral fragment, required double ligation. The upper fragment of the ulna was resected, as it could not be satisfactorily cleansed. Partial reduction of the various bony displacements was made, but no form of internal fixation was employed. Skiagraphs, made as soon as the patient could be moved from his room, showed that fairly good position of the bones had been secured. The soft parts healed with very little sloughing, no sequestration of the bones occurred, and firm union developed in the humerus within six weeks (Figs. 14 and 15). Though only fibrous union occurred in the forearm, and necessitated a subsequent operation, it is manifest that no attempt at primary fixation of the fragments would have obviated this result; while the absence of foreign bodies from the wounds permitted rapid healing of the soft parts, a contrast to the cases where metal splints are employed.

I may add that in spite of the extensive lesions of the soft parts in Cases I and II, there was never any disability from stiffness of the neighboring joints; the fingers are perfectly supple.

CASE III.—*Compound comminuted fracture of radius, and compound dislocation of ulna at wrist.*

William E., aged fifty-two years, had his left forearm caught in machinery, October 13, 1914, and was brought to the Episcopal Hospital. The carpal extremity of the ulna was sticking out through the soft parts for three or four inches, and there was a

Fig. 14

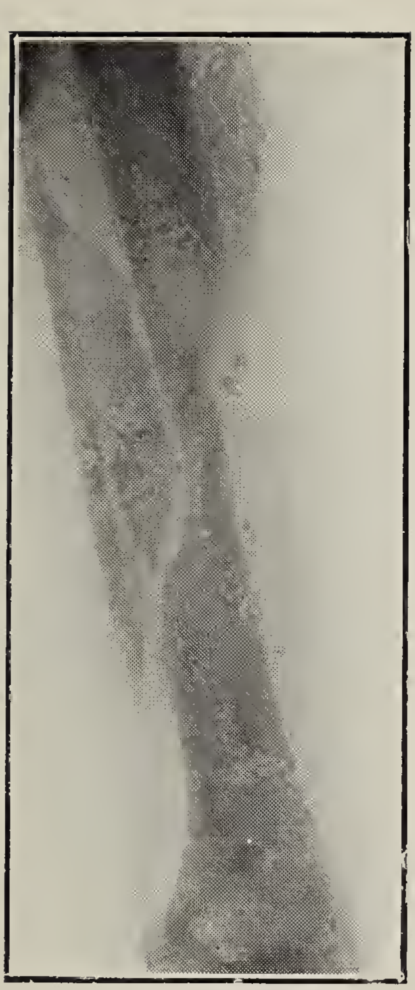
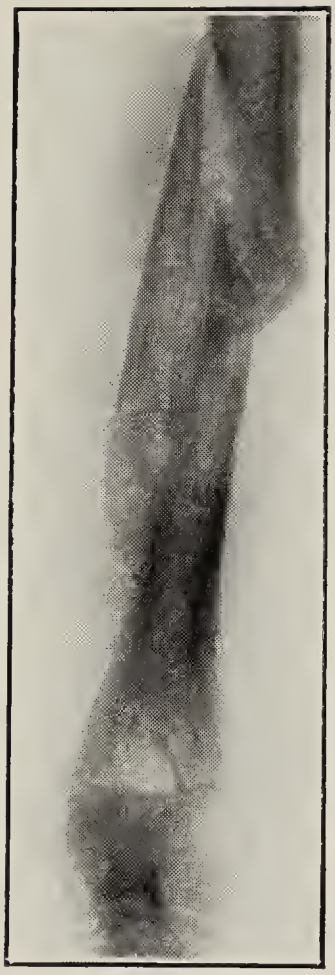


Fig. 15



Figs. 14 and 15.—Case II. Compound comminuted fracture of humerus, immediately after reduction by incision, and six months later

Fig. 16

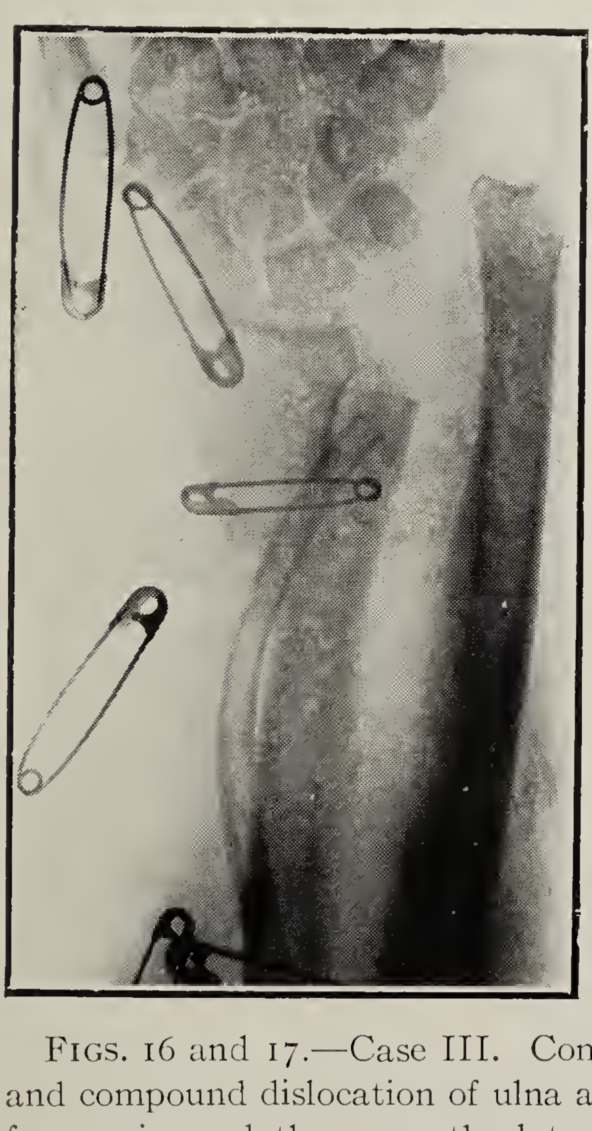
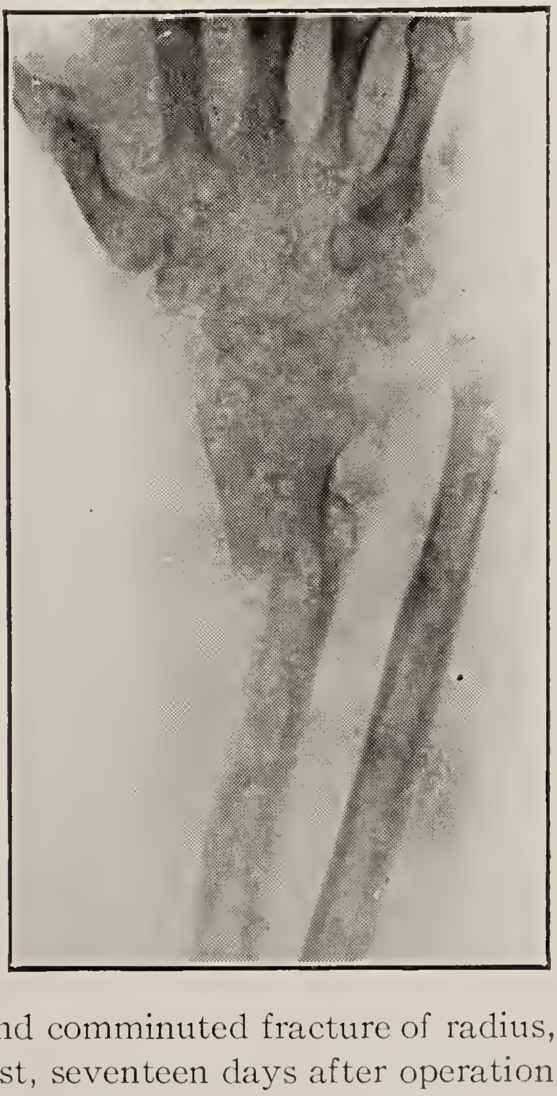


Fig. 17



Figs. 16 and 17.—Case III. Compound comminuted fracture of radius, and compound dislocation of ulna at wrist, seventeen days after operation for sepsis; and three months later. Note spontaneous improvement in position of bones.

Fig. 18

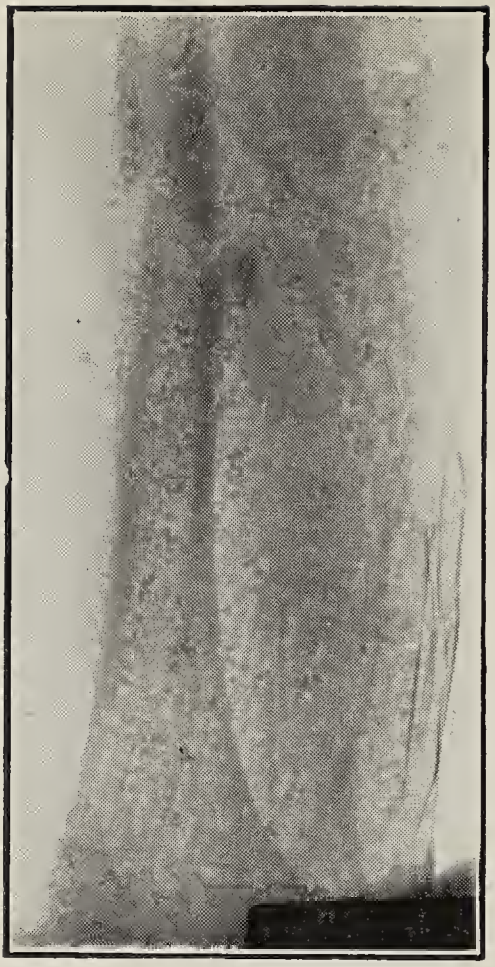
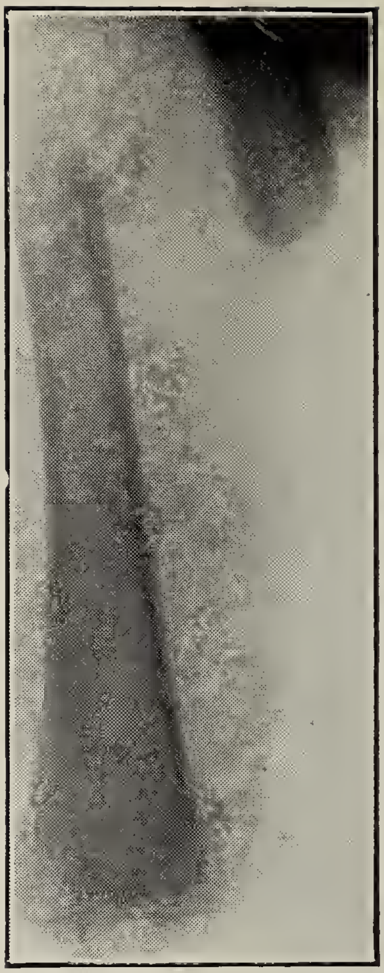


Fig. 19



Figs. 18 and 19.—Case V. Compound comminuted fracture of femur, anteroposterior and lateral views.

Fig. 20

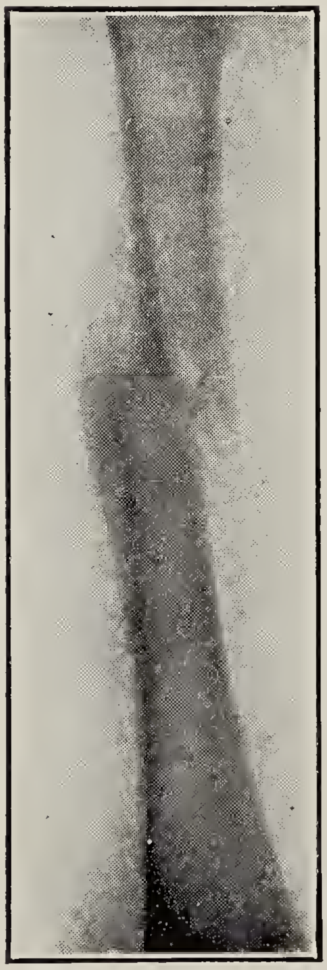
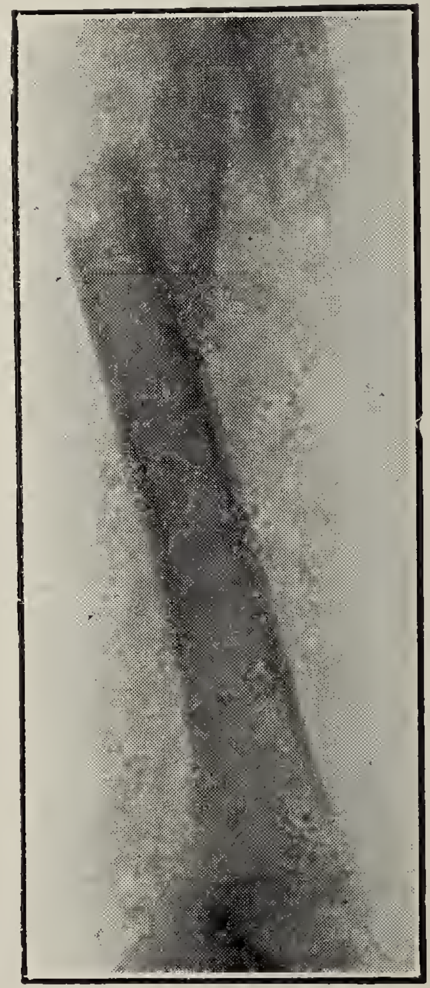


Fig. 21



Figs. 20 and 21.—Case V. Two months after injury, anteroposterior and lateral views.

compound comminuted fracture of the radius, with great laceration of the soft parts. After having his wounds dressed in the receiving ward, the patient absolutely refused to remain in the hospital, maintaining that the injury was trivial. Four days later, October 17, he presented himself at the dispensary, and was again urged to remain in the ward, but again went home. On his return home, however, he found himself so weak that he sent for the ambulance, which brought him to Dr. Frazier's service at 5.30 P.M.

*Operation.* At 7.30 P.M., I operated. The hand, forearm, and arm were greatly swollen; there were lymphagitic streaks extending toward the axilla, and the man was very septic, with a temperature of 101° F. All the sutures were removed, and the original wounds were enlarged, giving vent to large amounts of pus. The lower end of the ulna was again luxated, and was left protruding for about 7 cm. (nearly 3 inches), in order to provide for drainage; and a tube was passed through the wrist-joint, which was full of pus, and another tube across the forearm at the site of fracture, through the interosseous space. Culture of the pus gave a pure growth of staphylococcus aureus. All opened surfaces were swabbed with hot carbolic solution (1 to 20), alcohol dressings were applied, and the forearm was suspended vertically for a week. The patient's temperature fell at once, the swelling subsided within a couple of days, and by dint of absolutely disregarding the position of the bones, it became possible to save the patient's arm, which was in a condition almost justifying amputation when first seen. Fig. 16 is from a skiagraph made seventeen days after the operation; it shows the ulna still projecting far beyond the soft parts, and the radial fragments overlapping until the joint surface is at the level of the upper fragment. The ulna gradually was covered in by the soft parts, and two months after operation projected less than an inch. Fig. 17, from a skiagraph made nearly three months after operation, shows to what a surprising degree the radial deformity has been spontaneously corrected. On January 15, 1915, three months after injury, I resected the end of the ulna, and removed a few small sequestra from the radius, in which fibrous union was present. There was at this time scarcely any suppuration from the few sinuses still present, and active movements in the fingers were beginning to be possible.

This case shows the great danger of inefficient care of the soft parts from the first. Had the patient been willing to remain in the hospital when first injured, with his forearm in vertical suspension, and with irrigation, it is quite possible that no serious infection would have occurred; and that the soft parts would have been in a condition to warrant one in correcting the bony deformity before malunion had developed. As it is, the man put his life in jeopardy, very nearly lost his arm, has been totally incapacitated for months, and will never possess a very useful hand.

### 3. TREATMENT OF DISABILITY FROM LESIONS OF THE SOFT PARTS

Active movements and functional use of the limb are the most important factors in overcoming disabilities due to lesions of the soft parts; *massage* is a valuable adjuvant; but passive movements, especially if forced, usually are useless and often are harmful. These facts should be, and indeed actually are well known, but I see them ignored with lamentable frequency. To cite a case in point:

Case IV.—*Stiff elbow following forced passive movements after fracture.*

A surgeon brought his nephew to see me August 9, 1911. The boy, aged seven years, had injured his left elbow eight weeks previously. His uncle had dressed the elbow in the Velpeau position, and had a skiagraph taken, which showed a fracture of the external condyle of the humerus, not fully reduced. Three weeks after the injury the boy was taken to a consultant, who made further attempts, under ether, to reduce the deformity. The elbow was then dressed on an anterior right-angled splint for two days; then all dressings were discontinued, and four days after the attempted reduction passive movements were prescribed. This prescription was carried out most energetically by the masseur, who *forced* the elbow into full flexion and full extension *every day* for three weeks continuously. By this time the boy had suffered so much pain, and the elbow had become so manifestly worse, that a halt was called.

When I was asked to see the boy, eight weeks after the injury, and one week after passive motion had been stopped, I found the joint hot, tender, and painful on motion. The range of motion was only 50 degrees (85 to 135 degrees). The lower end of the humerus was half again as thick as normal in its anteroposterior diameter and there was tenderness on pressure over both biceps and triceps. Skiagraphs made the previous day showed the joint fairly normal, but there was new-formed subperiosteal bone on the front and back of the humerus, where the forced passive motion had exerted its greatest influence; while the only new-formed bone visible in anteroposterior view was above the fractured external condyle; but there was very little exuberant callus here.

My advice was to do absolutely nothing to the boy's elbow for a month, and to let him use it as he wanted. His uncle afterward reported to me that at the end of two months the range of motion in the elbow was very nearly normal in both flexion and extension, and that the joint gave no further trouble.

This lesson I learned early in my professional life. As a resident physician in the hospitals some of my chiefs "believed in the use of early passive motion" for fractures around the elbow; and many is the elbow on which at their instigation I enforced violent passive movement with the idea of preventing or of breaking up adhesions, destroying or wearing away exuberant callus, etc. The children kicked, screamed, and yelled; their parents, the orderly, and the nurse held them still, while I gave them excruciating pain, and unwittingly aroused more osteogenetic and inflammatory processes around the elbow than were present before; and I never saw an elbow which failed to stiffen up under this treatment. Fractures around the elbow I regarded as hopeless; I anticipated a stiff joint, deformity, or at least considerable limitation of motion almost in every case, and I rarely failed to find it. But as years have gone by I have become more and more convinced of the truth of Stimson's epigrammatic statement: "If you leave the arm alone, you save your time, and the patient's time, and he gets well quite as promptly."

Now what is true of the elbow-joint is true of all joints; and even in the case of fractures of the diaphyses of long bones *forced, painful, passive movements always are to be avoided.*

If the neighboring joints are stiff, encourage the patient to use the parts as much as he can; aid the absorption of exudate by hot-air baking and massage; but beware of passive motion.

It must be admitted, however, that occasionally in adults it is possible to improve the range of motion in a joint by forcible passive movements under an anesthetic; but it is possible only when the joint is no longer hot and painful, only when the inflammatory reaction initiated by the fracture has long since subsided.

## 4. FRACTURES IN THE DIAPHYSES OF THE LONG BONES

I come next to speak of the supposed necessity, or at least of the propriety of securing anatomical replacement of the fragments in a diaphyseal fracture. Not only is *such* replacement often impossible, but I do not hesitate to state that in most cases I regard it as totally unnecessary. Reasonably accurate reduction is desirable, and usually possible; but it is a very rare thing for disability to result solely from failure to secure anatomically accurate reduction of such a fracture. It is sufficient, in such fractures, as I have pointed out on former occasions, to secure *firm bony union*, with *no appreciable shortening*, and with *preservation of the axis of the limb.* The power of nature in rounding off bony prominences and in bridging gaps by callus is especially manifest in childhood; and it is in them that the most satisfactory results are obtained from unpromising beginnings. But this power, though less manifest in adults still is active in them. Let me cite several cases in illustration.

Case V.—*Comminuted fracture of left femur; irreducible; recovery without disability.*

Milton G., aged seven years, was admitted to Dr. Frazier's service in the Episcopal Hospital November 1, 1914. A wagon had run over the boy's thigh, and on account of the condition of the soft parts, attempts at reduction were delayed. There was 1.5 cm. shortening. Meantime the limb was placed in a Hamilton splint, and sufficient weights were applied to overcome shortening almost entirely. Skiagraphs, however, showed very marked displacement of the fragments (Figs. 18 and 19). On November 20 the child was etherized and I made attempts to secure reduction. Subsequent skiagraphs showed that the position was scarcely at all improved, and on account of the very wide separation of the fragments, I feared non-union, in spite of the patient's youth. Therefore I thought that operative reduction was indicated. But two days later, November 22, before operation could be done, the children's ward was placed under quarantine for an outbreak of scarlet fever. The limb was therefore redressed on a Hamilton splint, and conservative measures continued. On December 20, some union was apparent, and early in January union became very firm. There was now only 0.5 cm. shortening. The *vis medicatrix naturæ* had done its work well (Figs. 20 and 21). Toward the end of January, the boy was able to walk, and there is every prospect of his ultimate recovery of perfect function.

This case of course is an extreme example, but serves well to demonstrate the slight importance in children of accurate anatomical reduction of diaphyseal fractures. Let me now cite some cases in adults. One such is the fracture of the humerus (a double compound fracture) which occurred in the patient already recorded herewith as Case II. Other examples are the following:

CASE VI.—*Fracture of shaft of femur, unreduced for thirty years. Shortening* 6.5 *cm.; very little disability.*

A man, aged fifty years, came under my care in Dr. Frazier's service at the Episcopal Hospital in August, 1911. Thirty years previously he had sustained a fracture of his femur; he had just fallen again, and feared he had re-fractured the same bone. There appeared, however, to be only a contusion. For thirty years this patient had pursued his usual occupation, entailing hard physical labor, earning from three to four dollars daily, with shortening of 6.5 cm. He wore a lift in his heel, and walked without a disabling limp. He had some pain in his thigh in damp weather. I asked him how much he could have made if his thigh never had been broken, and he replied, perhaps five or six dollars daily (Fig. 34).

CASE VII.—*Fracture of left femur, unreduced; no disability.*

Louis M., aged fifty-five years, was admitted to Dr. Frazier's service in the Episcopal Hospital October 31, 1914, with compound fractures of both femurs. In the preceding May he had fractured his right femur, but the second fracture, in November, 1914, occurred a little below the previous injury. The skiagraph made after the second fracture (Fig. 22) shows that the first fracture had united with considerable deformity. In spite of this deformity, this patient had only 2 cm. shortening on leaving the hospital after his first accident. He had used crutches for five weeks after his discharge, being out of work thirteen weeks in all; and had worked as teamster until his second accident, without any disability whatever. His knee, he said, flexed to a right angle; he wore no raise on his heel to compensate for the shortening, and he considered himself an entirely able-bodied man until his second accident. No better proof of the solidity of the union could be required than is furnished by the fact that the second accident produced not a re-fracture at the site of the old injury, but an entirely distinct fracture below.

Of course the results in these patients do not form an argument against attempting to secure better reduction, but they certainly prove that very marked deformity (so long as the axis is preserved) in a diaphyseal fracture may cause very little disability. These cases are examples of extreme deformity; and if satisfactory function can be secured in such cases, much more certainly will it be secured in cases where the primary deformity is slight or in those in which reduction is easily obtained. Certainly I admit, and no one will dispute, that it is desirable to secure as accurate reduction as is possible; but I contend that sufficient reduction usually can be secured (almost always in children) without open operative intervention.[1]

## 5. FRACTURES NEAR JOINTS

Very different are fractures near joints. Quite apart from the questions of axial deviation, of rotatory deformity, of shortening, and of lesions of the soft parts (all of which factors must be considered here as well as in fractures of the diaphyses), there is the additional and all-important factor of *impairment of joint motion.* Unless accurate reduction is secured there will be permanent disability of some degree, varying with the extent of persistent deformity.

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[1] I may here emphasize the importance of the position in which the limb is dressed, as one of the most valuable aids to reduction. Weight extension, moreover, is applicable to fractures of the upper limb as well as to those of the thigh and leg, though this fact often is forgotten. I merely mention in passing the value of Steinmann nail extension in fractures of the leg, where the seat of fracture is too low for the ordinary Buck's extension apparatus to be efficient.

Fractures at the elbow and the ankle probably furnish the largest proportion of cases of disability and from the slightest grades of deformity; but fractures at the wrist are a frequent cause; while those of the shoulder and hip, though relatively less frequent, nevertheless present a rather high percentage of disability. Fractures at the knee (except those of the patella, which may be disregarded in this connection) are so rare that few such patients are seen complaining of disability.

It will be noted subsequently that *nearly all fractures which require operation for the relief of disability due to malunion of the bones are fractures near joints.* So convinced am I of the necessity of securing accurate reduction in joint fractures, that I believe open operative reduction should be undertaken in the vast majority of cases where bloodless attempts fail to secure anatomical reposition. Of course open treatment in these as in other fractures must not be adopted except where suitable facilites exist, both as regards the operator and the operating room.

Fortunately the bloodless reduction of joint fractures is relatively easy compared to that of diaphyseal fractures, since the position of the shorter fragment usually can be controlled through the neighboring joint. Thus, *abduction* at the shoulder and hip, *flexion* at the elbow and knee, and *adduction* at the wrist and ankle may be employed when necessary to maintain the fragment in apposition.

I believe it is much more important, also, in joint fractures than in diaphyseal fractures for reduction to be secured as soon as possible after the accident. Delay of three or four days may not only render reduction extremely difficult or even impossible, but may allow such swelling of the soft parts to occur as to ensure disability on this score even if reduction of the bones is subsequently obtained.

It is important also in joint fractures, when once accurate reduction has been obtained, to leave the joint at rest until bony union has occurred. Attempts at early passive motion are particularly harmful in these fractures. When once bony union has occurred, the patient will be able to execute active movements without pain, and in this manner will regain the normal range of motion in a short time. If stiffness persists, it is due to the condition of the soft parts; and this condition is due in its turn either to failure to reduce early enough and accurately enough, or to neglect of the soft parts while the fracture was mending. If the soft parts are in good condition and joint motion is persistently limited, it will be found that whatever range of motion exists is painless, and that arrest of motion at each end of the arc is produced by bony contact. Under such circumstances no increase in the range of motion can be secured except by operation (excision of callus, arthroplasty, etc.). If motion is painful, and it is not arrested by bony contact, improvement may be looked for as the condition of the soft parts returns to normal, under conservative treatment (active movements, light massage, hot-air baking, etc.).

### 6. THE PROPITIOUS TIME FOR OPERATION

It is important to determine as soon as possible after the injury whether or not operation will be required; and the operation, if required, should be undertaken neither too early nor too late. Except in the case of severe compound fractures, when the first dressing should assume the character of a formal operation, it is not desirable to operate before the fifth day. But it is possible, almost always, by the tenth day after the accident, to know whether or not operation will be required. This allows time for thorough examination and diagnosis, including the making of the necessary skiagraphs (always in two planes), as well as for attempts at reduction (under an anesthetic if necessary), and for the making of more skiagraphs to learn what has been accomplished.

Operations before the fifth day are more dangerous than those done between the fifth and tenth day, because the condition of the soft parts predisposes them to infection. It is best not to operate until sufficient time has elapsed for subsidence of swelling and commencement of organization of the effused blood. In the case of the femur this may require postponement of the operation for two weeks or even longer. And in any case where the skin is broken (as is frequently the case in fractures of the leg, even when there is not a true compound fracture), it should be the invariable rule not to operate until the skin is soundly healed.

On the other hand, an operation once determined on should not be unduly postponed. It is both more difficult and more dangerous to correct malunion or non-union by operation than it is to prevent them by the same means. If malunion occurs it is often impossible to overcome the shortening, and the most that can be done is to restore the axis of the limb, or to relieve the soft parts of injurious pressure by excision of exuberant callus, etc. Non-union, except in compound fractures, almost always is due to imperfect reduction of fragments when the fracture was recent. When operation is delayed beyond the second week, there is almost always found more or less osteoporosis of the distal fragment; this renders it difficult to make screws hold in it, and it is often necessary to reinforce the application of a plate by encircling wires.

Hence it is the duty of the modern surgeon to learn and to teach others which classes of injury require primary operation, and which do not. He should not start out with preconceived notions, that all fractures require primary operation, or on the other hand that primary operation is never necessary. Nor should he base his judgment on purely theoretical grounds. I believe the best and safest way to acquire knowledge on this subject is *to study the end-results of fractures*, and to work backward from them until we learn which fractures, and with what grades of primary and unreduced deformity, give us lasting disability, and which do not.

## 7. TREATMENT OF DISABILITY FOLLOWING DIAPHYSEAL FRACTURES

*If operative treatment is required for old diaphyseal fractures it is because of non-union or because of concurrent lesions of the soft parts, and scarcely ever because of malunion of the bones.* This statement embodies the conclusion I have reached by a study of end-results. In the patients who have come under my care for disability following diaphyseal fractures, the disability was clearly due to one of two factors: either non-union or lesions of the soft parts.

Malunion in a diaphyseal fracture, regarded as affecting the bone itself, may cause (1) shortening, (2) axial or rotatory deformity, or (3) non-union may be present. Neither malunion nor non-union of themselves will interfere with the free movements of the neighboring joints. Such limitation of motion, as well as persistent swelling, aches and pains, stiffness in wet weather, etc., almost without exception are due to concurrent lesions of the soft parts. I have cited already several examples to demonstrate the very slight disability which follows even considerable bony deformity, so long as the soft parts are in good condition. Shortening alone, even in the lower extremity, causes surprisingly little disability, so long as union is firm and the axis of the limb is preserved. Now, though shortening of 1 to 3 cm. is sufficiently common after non-operative treatment, axial and rotatory deformity are very rare, and scarcely need be considered as causes of disability.

Non-union is the chief factor which causes disability in cases of diaphyseal fracture; but it also is comparatively

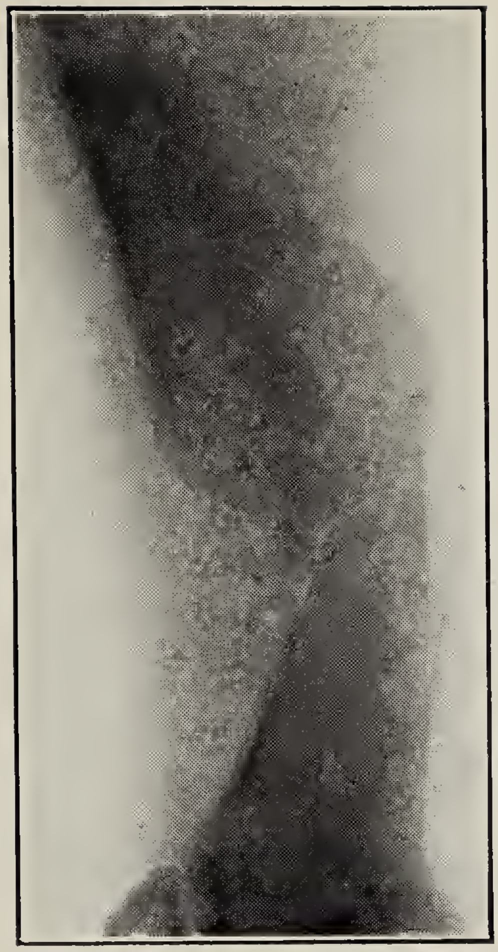


Fig. 22, Case VII.—Refracture just below point of malunion of old fracture.

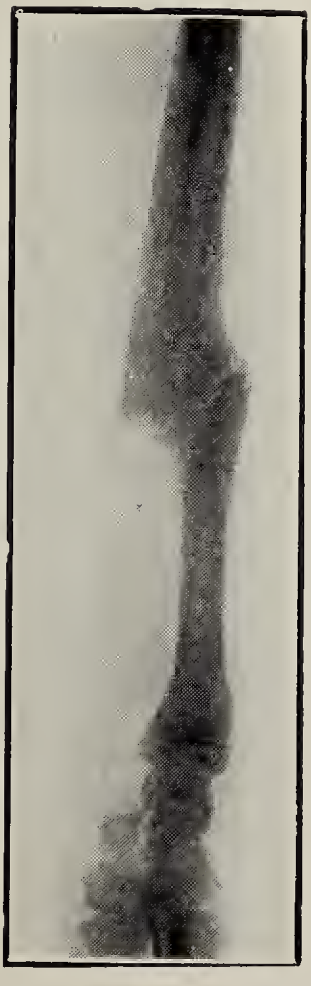


Fig. 23, Case VIII.—Malunion of radius and ulna with secondary paralysis of ulnar nerve.

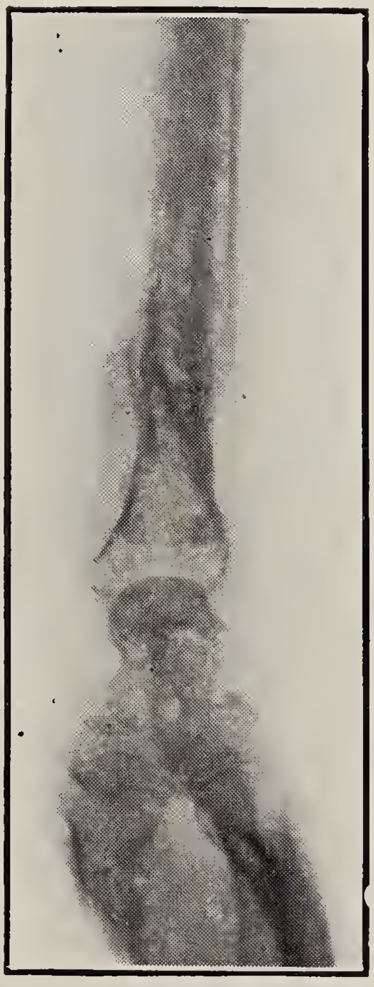


Fig. 24, Case IX.—Malunion of radius and ulna.

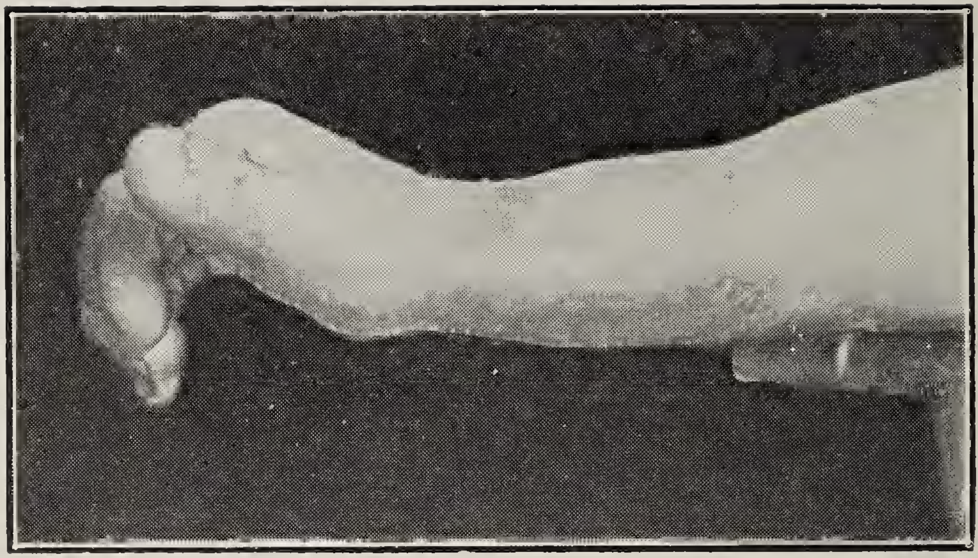


Fig. 25, Case IX.—Limited flexion of fingers before operation, with lost supination.

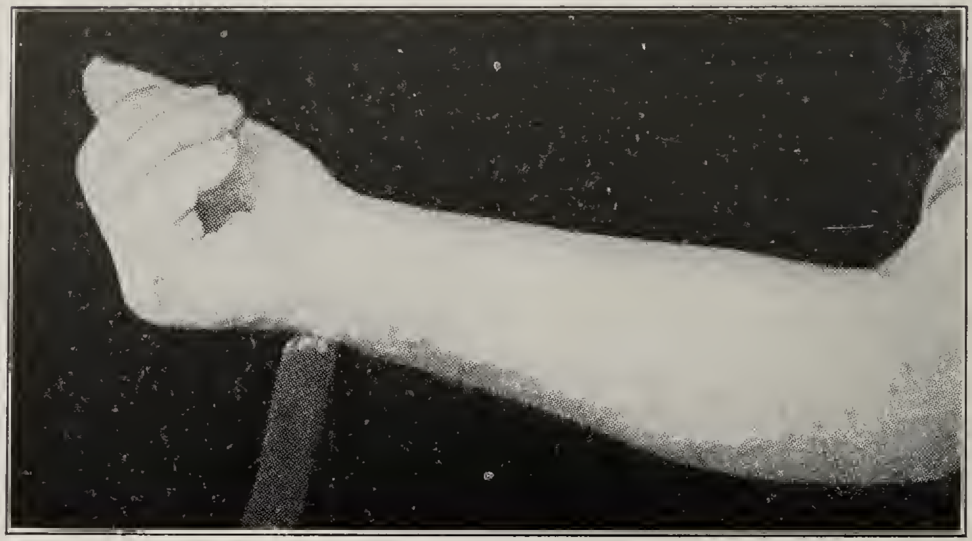
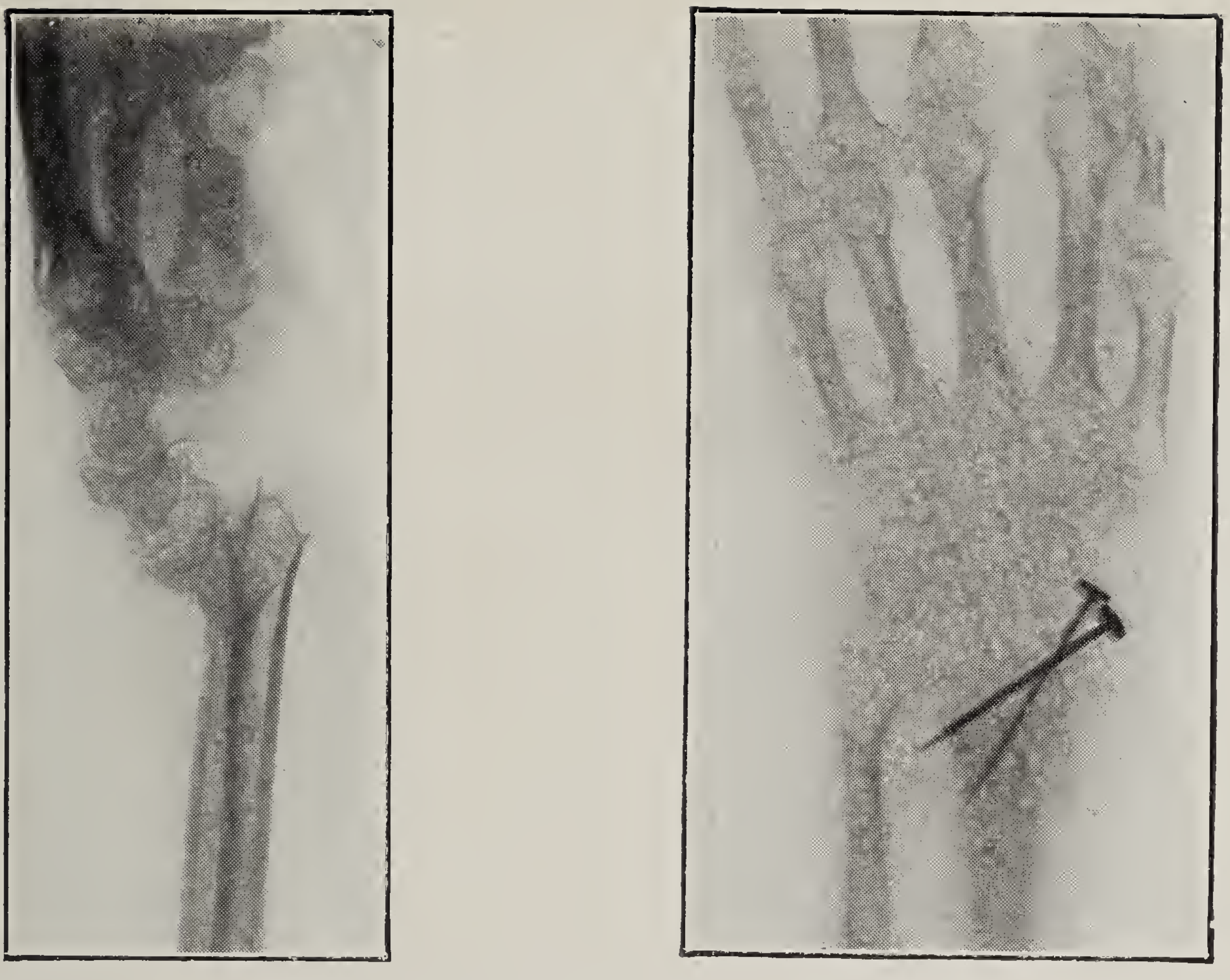


Fig. 26, Case IX.—After operation, able to make a better fist, and supination regained.

FIG. 27 FIG. 28



FIGS. 27 and 28, CASE X.—Malunion of a Colles' fracture, before and after operation.

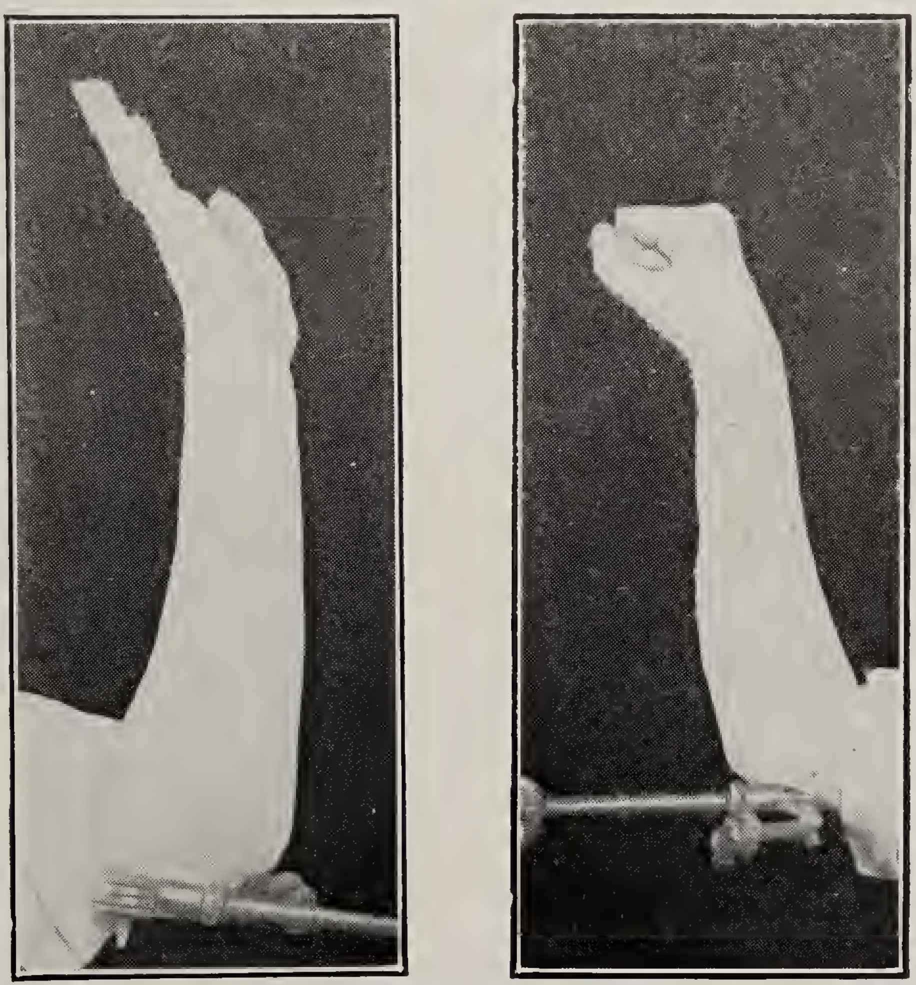


FIG. 29, CASE X.—Restoration of form and function after operation.

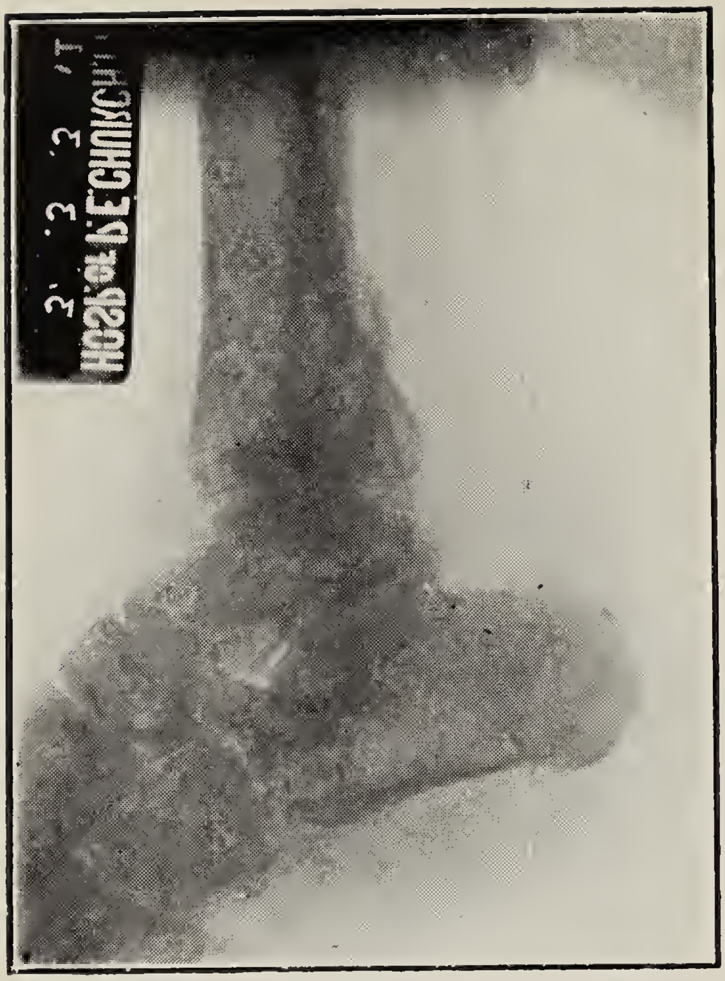


Fig. 30, Case XI.—Malunion of ankle fracture, lateral view.

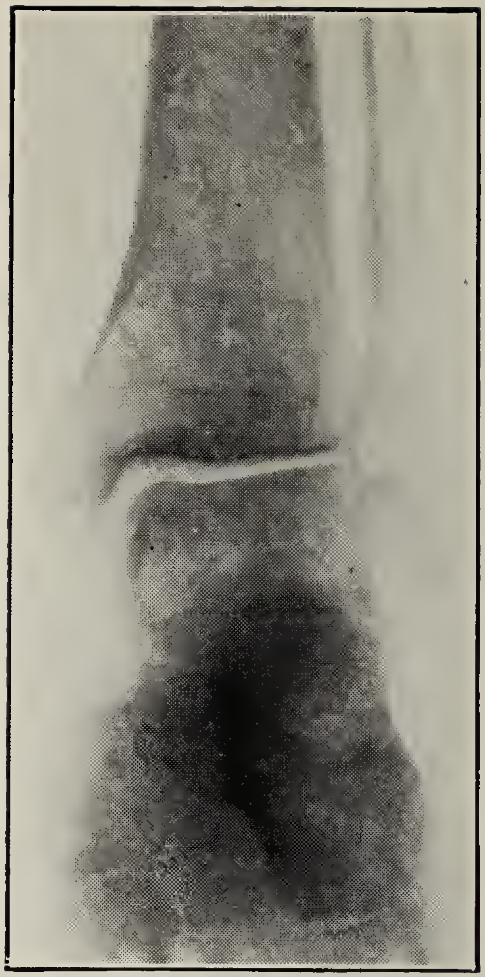


Fig. 31, Case XI.—Malunion of ankle fracture, anteroposterior view. Note diastasis of inferior tibiofibular joint.

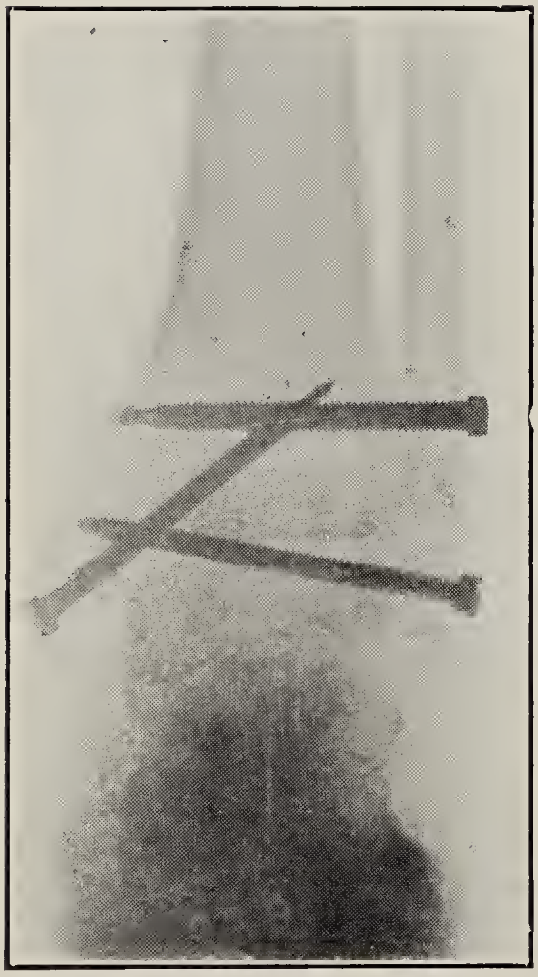


Fig. 32, Case XI.—After operation.

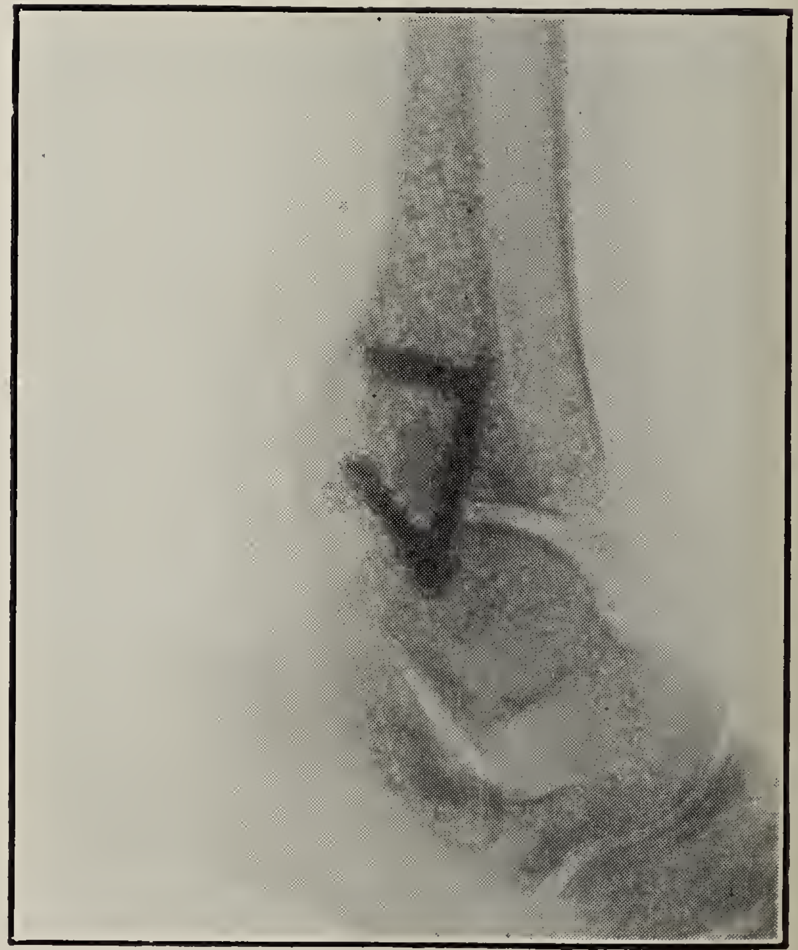


Fig. 33, Case XI.—After operation.

very rare. In all the recent fractures treated by myself, probably approaching a thousand in number, I have never yet had a case of non-union, except in a few cases of severe compound fracture in which immediate operation was required on account of the condition of the soft parts (Cases I and II are examples), and in some cases of fracture of the neck of the femur in the aged.

On the other hand, I have seen numerous cases in which lesions of the soft parts were the cause for which the patients sought relief.

CASE VIII.—*Malunion of fracture of radius and ulna; paralysis of ulnar nerve.*

Dolores D., aged thirteen years, was referred to my service at the Episcopal Hospital November 16, 1914, by Dr. E. E. W. Given. On August 6, 1914, this girl had sustained a fracture of both bones of the left forearm; four weeks after the splints had been removed, and eight weeks after the first accident, she fell again, but did not completely re-fracture the bones. Splints were re-applied for a further period of about four weeks, but after their removal pain and disability persisted, and considerable bony deformity was present. When the child first came under Dr. Given's notice, in November, more than a month after the second injury, there was readily visible bony deformity, and the hand was not only useless, but the seat of constant pain. There was anesthesia and hyperesthesia in the distribution of the ulnar nerve, with wasting of the thenar and hypothenar eminences and of the interosseous muscles.

At operation, November 25, 1914, the ulnar nerve was found caught in scar tissue over the lower end of the upper ulnar fragment (Fig. 23). Union in both radial and ulnar fractures was only fibrous, and resection and plating of both bones was done. Within three days there was evidence of some return of sensation in the ulnar distribution, and by the end of a week sensation was normal. Progress thereafter was uneventful. At the end of four weeks firm union was present, the soft parts had returned to normal, and a fair range of rotation was present. Eventually recovery was complete.

It appears to me that if proper care had been taken of the soft parts, bony union would have occurred, even after the second injury, in spite of the deformity present; and that this deformity alone would not have been a cause of much disability. Opportunity being afforded for operation, because of the nerve lesion, I believe I was justified in securing better reduction of the fragments.

CASE IX.—*Malunion of fracture of radius and ulna; disability from adhesions of the soft parts.*

Joseph P., aged forty-three years, was referred to my service at the Episcopal Hospital in February, 1914. Two months previously his hand and forearm had been caught in a cable, fracturing the radius and ulna above the wrist, and greatly contusing the soft parts. The fractures had united with scarcely any axial displacement, though the lower fragments were displaced toward the extensor surface (Fig. 24). Wrist motion was greatly limited, and though rotation of the forearm was possible through more than half the normal range, the hand was perfectly useless, owing to inability to make a fist (Fig. 25). For more than six months this man was treated by massage and passive motion, with neglible improvement. He then consented to an operation, which was done August 8, 1914, and consisted simply in chipping off the bony angles and thoroughly freeing the adherent muscles, back and front. The result was restoration of normal rotation, and very nearly normal movements in the wrist. But it was still impossible for him to make a fist, owing to persisting ahdesions in the hand and fingers. Finally, on November 11, 1914, he was again etherized, and these adhesions forcibly ruptured, and the fingers dressed in full flexion. This could not be maintained, however, and when last seen, early in December, 1914, he was still unable to make a tight fist, and therefore to hold a trowel and resume his former work as plasterer, though the condition was probably 75 per cent. better than before operation, and he could still make a living at other work (Fig. 26).

In this patient the deformity was by no means so great as to produce disability by itself; and if it had not been for adhesions of the soft parts, I have no doubt that an excellent functional result would have been secured without operation.

## 8. TREATMENT OF DISABILITY FROM FRACTURES NEAR JOINTS

When a fracture occurs close to a joint, very slight deformity may entail great disability. In the first place it is more important here than in the shaft of a long bone to maintain the axis of the fragments, since less compensatory bone changes can occur when one fragment is very short than when both are of nearly equal length. A slight tilt of the short joint fragment will cause marked deviation of the articular surface. I have already emphasized, perhaps sufficiently, the importance of accurate reduction in these lesions, and have cited cases indicating that in most instances such reduction can be obtained without resort to operation; but I cannot refrain from quoting some further examples which illustrate the very serious disability which follows neglect of accurate reduction.

CASE X.—*Malunion of a Colles's fracture of the radius; median neuritis; resection and screw fixation.*

Anna K., aged twenty-eight years, sustained a Colles's fracture of the right radius June 1, 1914. She was treated by her family physician, who placed the forearm on a straight palmar splint, but made no attempt to "set" the fracture. Over fourteen months after injury this young woman came under my care, complaining not only of the conspicuous deformity (Fig. 27), but of complete disability, from weakness, loss of flexion at the wrist, and sweating and paresthesia in the distribution of the median nerve. She was admitted to my service at the Episcopal Hospital, and I operated October 10, 1914. The carpal fragment was found united by bone to the extensor surface of the diaphysis, while the fractured surface of the diaphysis, free of all bony contact, jutted up against the median nerve and flexor tendons (Fig. 27). Resection and screw fixation restored the parts to normal (Fig 28), and on December 12, one week after her return to her usual work in a mill, the grateful patient returned to report that she found her hand in every way as good as it had ever been before the accident (Fig. 29).

CASE XI.—*Malunion of fracture at ankle. Resection and screw fixation.*

James C., aged thirty-one years, fractured his left ankle in July, 1914. In September, 1914, he came under my care in the orthopedic service at the Episcopal Hospital, because of persistent disability. He was unable to dorsiflex the foot beyond 90 degrees, and as a consequence had to walk with his whole limb in external rotation, carrying the foot in the coronal plane of the body. Even thus he suffered constant pains along the fibula from the external malleolus upward. A skiagraph (Figs. 30 and 31) showed an unreduced oblique fracture of the lower fifth of the fibula, entering the ankle-joint and detaching also the adjacent surface of the tibia. The astragalus was dislocated slightly backward, with the foot, and there was distinct diastasis at the tibiofibular joint, the fibula being abnormally movable on the tibia in the anteroposterior plane. A fracture of the internal malleolus was not visible in the skiagraph.

I kept this man under observation for two months hoping that improvement might occur; but he persistently urged operative treatment, on the ground of absolute disability for his former work. Finally, on December 9, 1914, five months after the fracture, I operated, resecting the line of fracture in the fibula, cutting a new groove for it in the tibia, detaching the misplaced internal malleolus (which was now seen to have been fractured) and restoring it to its normal position. The fragments were fixed by three large Lambotte screws. Figs. 32 and 33 show that the posterior displacement of the fibula and astragalus has been overcome, and that the fibula is fixed firmly against the tibia.

Not until eight weeks after operation was this man allowed to bear any weight on his limb. He was then able to walk painlessly and without further disability.

In these and similar cases the proper time for operative reduction of the deformity (if bloodless reduction was impossible) was while the lesions still were recent, and not after malunion had occurred, and the patients had been disabled for months.

## 9. TREATMENT OF NON-UNION

As I have already indicated, comparatively few cases of real non-union have come under my care. The distinction between delayed union and non-union may be said to be that, in the former, union will occur eventually without operative interference; while in cases of true non-union the bones remain ununited in spite of all methods of conservative treatment. I think this distinction is important to bear in mind, and believe that the surgeon should not be inclined to rush his patient with delayed union into a possibly very brilliant but by no means necessary operation. I am sure that in a good many of the cases of operative treatment of so-called non-union now being reported in medical literature, non-operative measures might very well have been effectual in securing bony union, and in as short a time as by operative treatment. I have especially in mind just now delayed union in fracture of the leg; I know of a case of this kind where bony union had not developed at the end of six weeks, and the surgeon at once resorted to bone transplantation. The operation was perfectly successful, and firm bony union developed. But then I recall numerous similar cases of delayed union where the patients developed bony union within a few weeks after being allowed to walk around in their gypsum cases. Of course if there is marked deformity in a case of delayed union, a better end-result usually will be secured by operative means, since reduction of the deformity can be obtained at the same time that bony union is encouraged.

But the surgeon must nevertheless stop to consider whether the patient's condition is not good enough as it is, even though firm union is not present. He should remember that bony union does not necessarily follow operation, and that operation may even make the patient's condition worse.

There appears to be little doubt that the most efficient way to secure bony union is to insert a bone transplant. I have recorded elsewhere (*Annals of Surgery*, 1914, ii, 779) the very satisfactory result obtained by this means in a case of non-union of the neck of the femur, in a young adult who had been unable to walk without crutches for eight months; and I have also employed it in the forearm. In young children, where the osteogenic powers are so much better than in adults, I prefer, when non-union occurs in a joint-fracture (I have operated on three of the external condyle of the humerus), to fix the fragments with sutures or screws, after freshening their surfaces.

## CONCLUSIONS

The conclusion of the whole matter seems to me to be a recognition of the fact that it is necessary and always will be necessary, for a large proportion of the profession, who are not trained surgeons, to have cases of fracture under their care. They cannot avoid it. They cannot adopt operative treatment even when it is requisite to prevent disability. The only remedy is to have them so well trained in measures that they can apply, as to enable them to secure the least possible degree of disability.

Then I believe the average surgeon should recognize that the operative treatment of fractures is not as indispensable as some writers would have us believe; and that he should continue in the future, as he has in the past, to give preference to non-operative methods; that he should feel prouder of curing ten patients without disability and without resort to operation, than of recording fifty or one hundred brilliant operations, most of which were really quite unnecessary had he only known enough surgery to make efficient use of non-operative means.

It is important also for the trained surgeon in charge of a case of fracture to be capable of recognizing while the lesion is still recent, whether or not open reduction is requisite. That cases requiring open treatment exist, even the utmost conservatism cannot deny; cases in which it is manifestly impossible to secure any sort of respectable result without operation.

Finally, may I venture to suggest that the patient's interests might perhaps be promoted, if not only the general practitioner but the average general surgeon as well should feel a little more Hallerian diffidence than he does at present about undertaking operations which may have (and frequently have had) such disastrous results as those for fractures of the limbs. Whether a patient will recover, or whether he will die, after such an operation, cannot always be foretold. A very slight lapse in aseptic technique may prove fatal. At all events, there is often a question whether as good a result might not have been obtained without operation. I often recall in this connection that wise maxim of Heister; it may be interpreted either of good or of bad results: *In prœdicendis fracturarum eventibus multa utique chirurgis opus est circumspectione.*

# FRACTURES OF THE FEMUR WITH END-RESULTS IN 62 CASES[1]

By CARL R. STEINKE, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

THE poor functional and anatomical results in fractures of the long bones of the extremities led the American Surgical Association and the British Medical Association to investigate the causes for such results. A waning interest in fractures has been renewed, and with the form-blanks covering all the important facts to be obtained concerning fractures, more uniform, complete and exact data are being gathered.

Much has been written concerning fractures of all bones, but the femur presents some of the greatest difficulties to be encountered, while its anatomical and physiological importance accentuates the necessity for a good result.

In considering fractures of the femur it is well to take them up in different groups according to the portion of the bone fractured, such as those of the upper end (neck and through the trochanters), the shaft and the lower end. The varieties, manner of production and treatment of fractures have been so thoroughly dealt with in the special fracture number of the *American Journal of Surgery* for March, 1915, by such authorities as A. Lane, W. L. Estes, A. P. C. Ashhurst, F. H. Albee, J. B. Walker and others, that they will not be considered in this paper except as they concern reported cases.

With the more general use of the Röntgen rays we are enabled to see the exact position of the fragments before and after treatment, so we can follow cases to observe how kind nature is in producing generous callus to cover our defective reductions, with resultant solid union (Figs. 34, 35, 36, 37, 38, 39, and 40). The anatomical relation of the fragments may be bad and still give a good functional result (Table IV).

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[1] Read by invitation before the Philadelphia Academy of Surgery, May 3, 1915. Reprinted from Annals of Surgery, November, 1915.

The basis of this article consists of the fractured femurs from Dr. Charles H. Frazier's service at the Episcopal Hospital during the 10 years from 1905 to 1915, and I am indebted to him for the privilege of reporting the cases.

Of the 109 cases on record we were able to follow 62 (57 per cent.); 15 fractured through the neck, 6 through the trochanters, 36 of the shaft, and 5 of the lower end. A number of the cases have been previously reported more in detail before this Academy and elsewhere by Dr. A. P. C. Ashhurst.[1] Of the 47 fractures not followed there were 4 each through the trochanters and of the lower end, 15 of the neck and 24 of the shaft; the condition of the patients at the time of discharge was as follows: 10 cured, 24 recovered, 10 improved, 1 at own request, and 2 refused treatment.

Considering the fractures of the neck there were 10 females and 5 males, with ages ranging from 14 to 84 years (average 64½). All were simple fractures produced by indirect violence. The time in the hospital ranged from 2 to 29½ weeks (average 6⅔), while the average time from work of those who survived was 22½ weeks. Of the 8 cases dying in the hospital (5 intracapsular) 6 were females and 2 males, with ages ranging from 65 to 84 years (average 74½). The cause of death was given as 1 each from uræmia, senility and carcinoma of the stomach (autopsy confirmation), 2 from pneumonia, while in 3 it was not stated. A peculiar fact is noted in that 6 of these occurred during the services of 1905 and 1906, but no definite explanation for this was found. The functional and anatomical results are given in Table I.

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[1] Ashhurst, A. P. C.: Fractures Through the Trochanters of the Femur, Annals of Surgery, Philadelphia, October, 1913, p. 495; also, The Prevention and Treatment of the Disabilities Following Fractures of the Limbs, Amer. Jour. of Surg., 1915, xxix, 114.

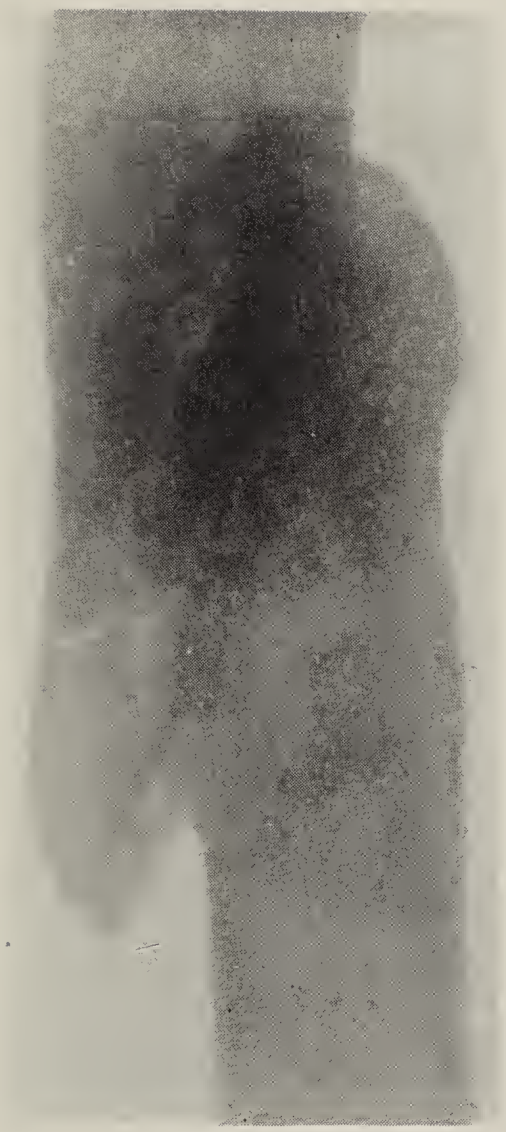

FIG. 34.—J. F., aged fifty years, who had broken his left femur shaft thirty years previous to this $x$-ray. He has two and one-half inches shortening, wears a high shoe and makes good wages, but says he could make one-third more if not for his disability. He has some pain in region of fracture during damp weather. Plate shown to illustrate the way nature cares for defects, giving moderate functional result. (Ashhurst.)

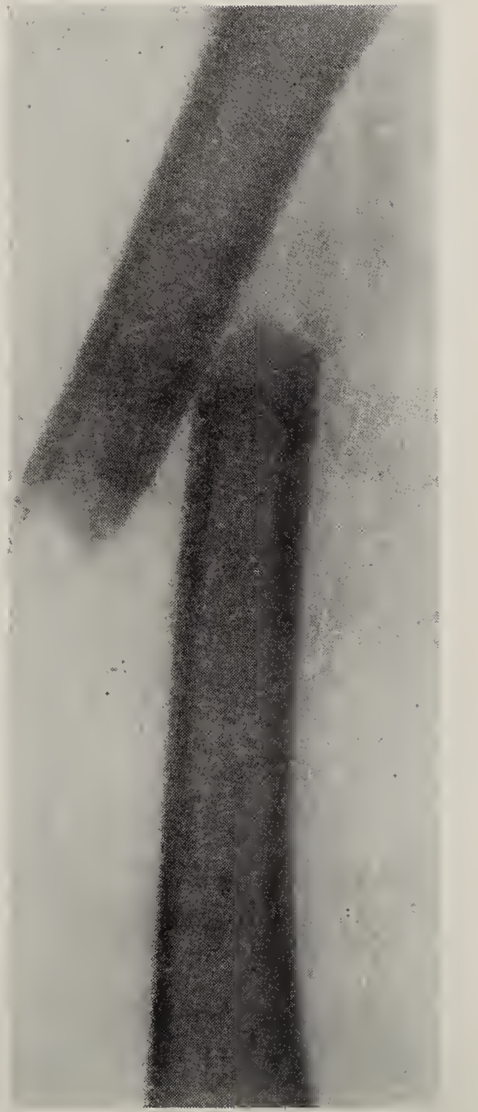

FIG. 35.—Case 11 of Table IV under Fractures of the Shaft, which was compound. E. F., girl seven years of age. Lateral view before setting under ether. Had 3 cm. shortening. Left femur. (Ashhurst.)

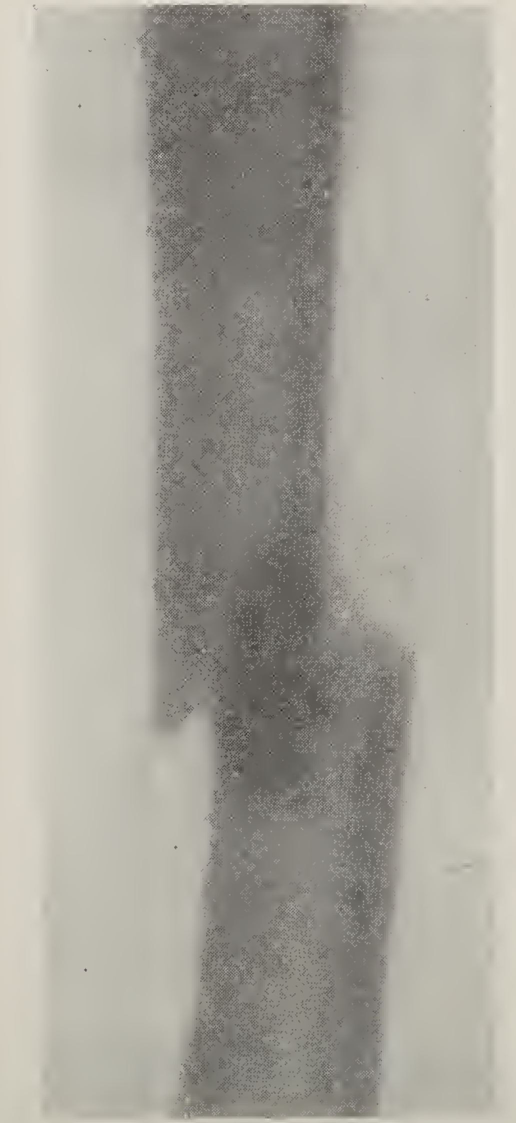

FIG. 36.—Same case as Fig. 35. Anteroposterior view before setting under ether.

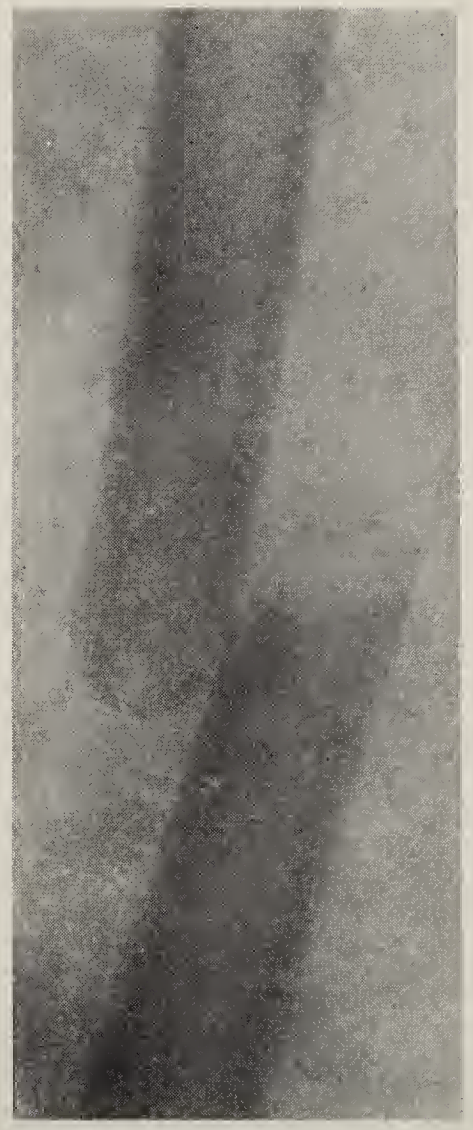

FIG. 37.—Same case as Fig. 35. Lateral view after setting under ether.

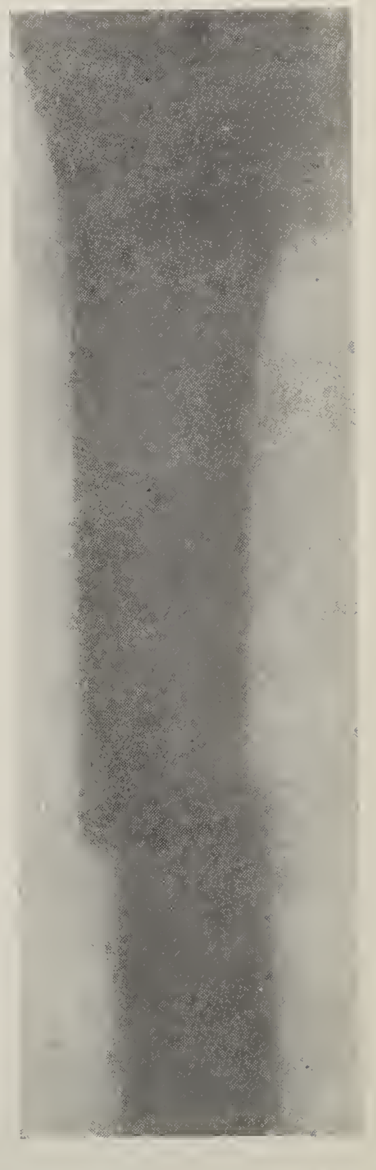

FIG. 38.—Same case as Fig. 35. Anteroposterior view after setting.

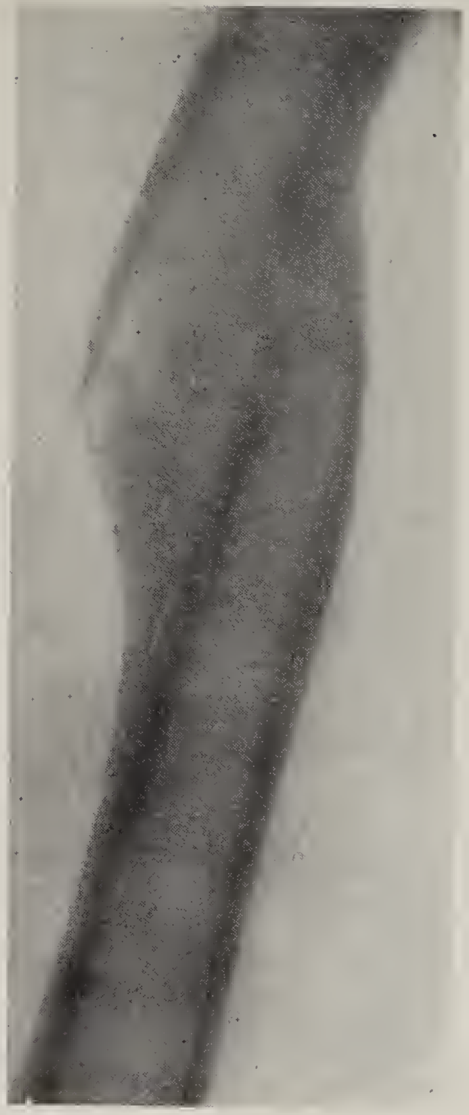

Fig. 39.—Same case as Fig. 35. Lateral view six months after setting.

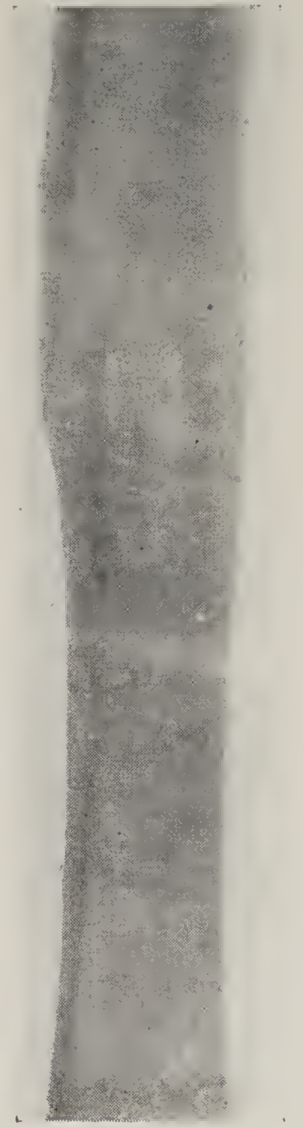

Fig. 40.—Same case as Fig. 35. Anteroposterior view six months after setting.

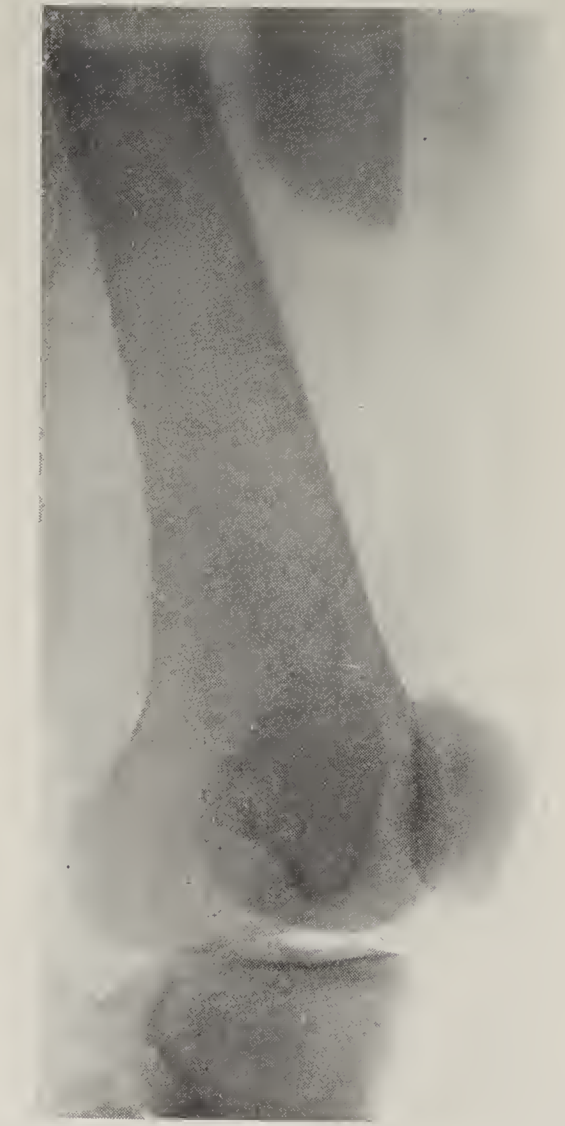

Fig. 41.—G. O'N., twenty years of age, fracture of femoral shaft. Lateral view.

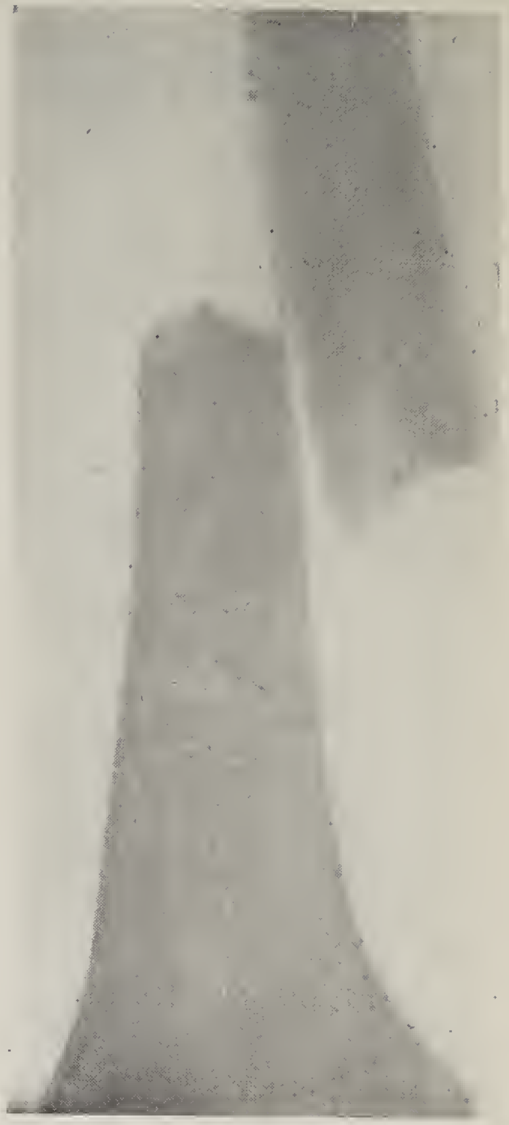

Fig. 42.—Same case as Fig. 41. Anteroposterior view.

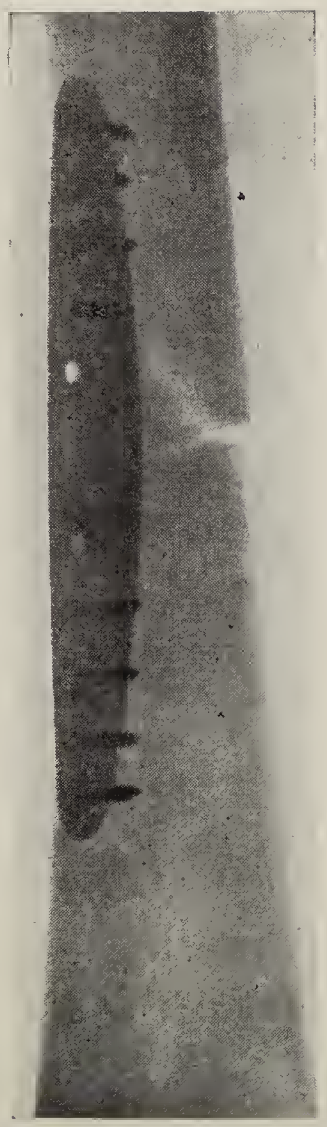

FIG. 43.—Same case as Fig. 41. Dr. C. H. Frazier's service, Episcopal Hospital. Operation by Dr. A. P. C. Ashhurst, November 17, 1914, with good result. *X*-ray of limb four weeks after Lambotte plate was applied.

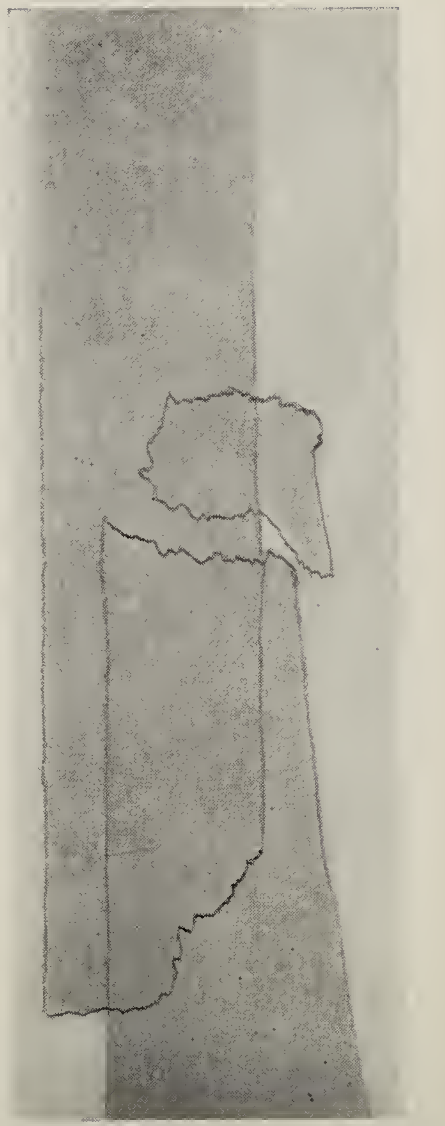

FIG. 44.—F. R., twenty-six years of age. Fracture of femur Dec. 5, 1909.

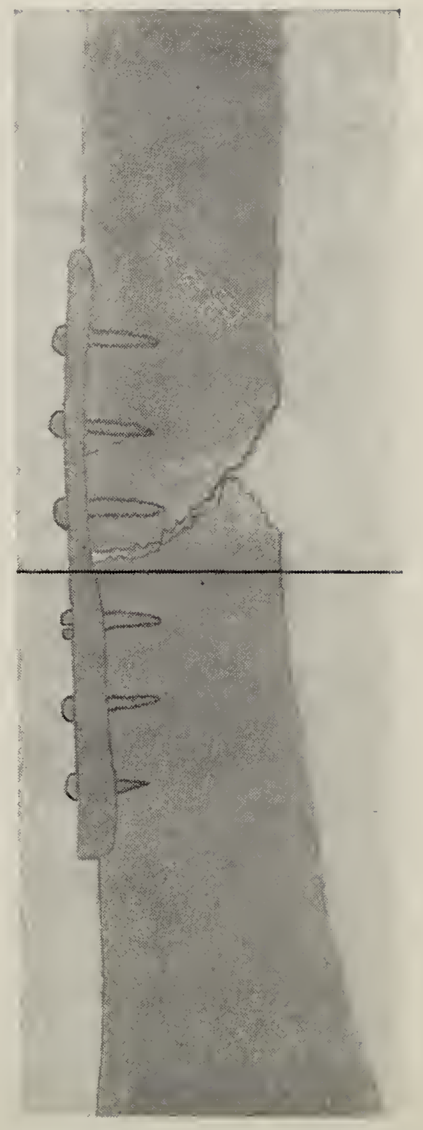

FIG. 45.—F. R. Same as Fig. 44, six weeks after operation (late) by Dr. Ashhurst.

There were 6 cases of simple fracture through the trochanters (Table II), all being of the male sex and treated non-operatively. Three were caused by direct and 3 by indirect violence. The number of weeks in the hospital averaged 7, with extremes from 4 to 10, while the time from work averaged 34 weeks, varying from 12 to 93. The earning power was less in 4 cases, each having a moderate anatomical result with moderate to bad functional result, while the 2 with the same earning capacity had both good anatomical and functional results.

Of the 5 cases where fracture occurred at the lower end of the femur, again they were all of the simple type produced by direct violence, all males with ages ranging from 6 to 61 years (average 34). One case with bad position of the fragments was plated with resultant good apposition but only moderate return of function. Here the time in the hospital varied from 2 to 11 weeks (average 5½), the patient going out in 2 weeks had the limb in a plaster cast returning later for the removal of the cast. The earning capacity in these cases was the same in 2 and less in 2; the fifth developed delirium tremens and died after 3 weeks. Functional and anatomical results are shown in Table III.

Over half the cases followed were fractures of the shaft, 33 males and 3 females with ages from 3 to 74 years (average 23⅓). Five of the cases were plated because of the inability to obtain proper reduction; 3 good and 2 moderate anatomical results followed, and 4 good and 1 moderate functional result. The time in the hospital ranged from 1 day to 59 weeks (average 10 weeks). One case died of shock the day of admission, while another died on the third day of a fractured skull. The disability period was between 8 and 115 weeks (average 16.9), giving 29 good, 4 moderate, and 2 bad functional results from 25 good, 6 moderate and 4 bad anatomical results. The case who stayed in the hospital 59 weeks and was out of work 115, developed delirium tremens, got the leg in bad condition, causing sloughing of tissue, when a cast was applied, with both bad anatomical and functional results. Thirty cases were of the simple type, while Cases 3, 9, 19, 26, and 27 were comminuted, and Case 11 compound, the fracture having been produced by indirect violence (see Figs. 35 to 40). Twenty-four fractures resulted from direct and 12 from indirect violence.

Tables V, VI, VII, VIII, and IX have been compiled so that the various summaries of results, such as age groups, disability periods, etc., of the different classes of fractures may be noted at a glance.

As to treatment the non-operative gave as good results, functionally, as the operative, although the plated cases were selected, which without operation most certainly would have given poor results. Anatomical results were based on röntgenograms when they were employed and in the other cases on the amount of shortening or deformity. The latter is faulty in that good approximation and alignment of the fragments is not necessary for a straight and unshortened limb. The skiagraph is the only accurate basis for our conclusions, but unfortunately not all had röntgenograms after completed treatment.

The mortality of 11 cases was not due directly to the fracture of the femur excepting in the one dying from shock, while in a number of the others it was a contributing factor.

In conclusion from the facts gathered:

1. Fractures of the neck were more frequent in the aged of the female sex, indirectly cause a high mortality because of the age of the patient and the lowered resistance, and give a moderate disability period.

2. Fractures through the trochanters were all males and gave a long disability period with lowered earning capacity in two-thirds of the cases.

3. Fractures of the lower end of the femur gave an average disability of about 6 months while a good functional result was obtained in about one-half of the cases. They were all males.

4. Fractures of the shaft gave the best results and had the shortest disability period. They were the most frequent and were mostly males.

5. The mortality from fractures of the femur other than of the neck was low, being about 2 per cent. in this series.

TABLE I.—FRACTURES OF NECK OF FEMUR

| No. | Sex. | Age. | Treatment. |  | X-ray. |  | Weeks in hospital. | Weeks from work. | Earning power. | Functional result. |  |  | Anatomical result. |  |  | Died. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Operative. | Non-operative. | Early. | Late. |  |  |  | Good. | Mod. | Bad. | Good. | Mod. | Bad. |  |
| 1 | M | 14 | .. | C | * | * | 6 | 10 | Same | * | .. | .. | * | .. | .. |  |
| 2 | M | 30 | .. | † | * | .. | 4 | 30 | Less | .. | .. | * | .. | .. | * |  |
| 3 | F | 58 | .. | † | * | * | 29½ | Idle | Idle | .. | * | .. | .. | * | .. |  |
| 4 | F | 60 | .. | E and L | .. | .. | 6 | 24 | Same | * | .. | .. | * | .. | .. |  |
| 5 | M | 63 | .. | E | .. | .. | 11 | Idle | Idle | * | .. | .. | .. | * | .. |  |
| 6 | M | 65 | .. | † | .. | .. | 2½ | .. | .. | .. | .. | .. | .. | .. | .. | Pneumonia |
| 7 | F | 67 | .. | E | .. | * | 6½ | 26 | Same | * | .. | .. | * | .. | .. |  |
| 8 | F | 70 | .. | E | * | .. | 17 | .. | .. | .. | .. | .. | .. | .. | .. | Uremia |
| 9 | F | 70 | .. | † | * | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | Cause not stated |
| 10 | F | 74 | .. | C | * | .. | 2½ | .. | .. | .. | .. | .. | .. | .. | .. | Carcinoma of stomach |
| 11 | F | 75 | .. | E and C | .. | .. | 3 | .. | .. | .. | .. | .. | .. | .. | .. | Senility |
| 12 | F | 76 | .. | † | .. | .. | 4 | Idle | Idle | .. | * | .. | .. | * | .. |  |
| 13 | F | 77 | .. | † | .. | .. | 6 | .. | .. | .. | .. | .. | .. | .. | .. | Cause not stated |
| 14 | M | 82 | .. | E | .. | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | Cause not stated |
| 15 | F | 84 | .. | † | .. | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | Pneumonia |
| Total 15 | M F<br>5 10 | Ave. 64½ | 0 | 15 | 6 | 3 | Ave. 6⅔ | Ave. 22½ | Same 3<br>Less 1<br>Idle 3 | 4 | 2 | 1 | 3 | 3 | 1 | 8 |

NOTE.—Case 3 died 3 years later. Case 5 died 5½ years later.

Key to tables:

Mod = Moderate.
E = Buck's extension.
L = Lateral extension.
VE = Vertical extension.
C = Plaster-of-Paris cast.
H = Hamilton splint.
S = Splint.
Splint = Coaptation splint.
SB = Sand bags.
† = Type not stated.
Idle = Not working, being too old or for other cause.
* = Time $x$-rays were taken and end-result in each case.

## TABLE II.—FRACTURES THROUGH TROCHANTERS

| No. | Sex. | Age. | Treatment. |  | X-ray. |  | Weeks in hospital. | Weeks from work. | Earning power. | Functional result. |  |  | Anatomical result. |  |  | Died. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Operative. | Non-operative. | Early. | Late. |  |  |  | Good. | Mod. | Bad. | Good. | Mod. | Bad. |  |
| 1 | M | 42 | . . | S and C | * | . . | 8 | 27½ | Less | . . | * | . . | . . | * | . . |  |
| 2 | M | 45 | . . | E | * | . . | 6½ | 12 | Same | * | . . | . . | * | . . | . . |  |
| 3 | M | 48 | . . | E | * | . . | 4 | 48 | Less | . . | * | . . | . . | * | . . |  |
| 4 | M | 54 | . . | E | * | . . | 4½ | 9½ | Same | * | . . | . . | * | . . | . . |  |
| 5 | M | 54 | . . | E and S | * | . . | 10 | 93 | Less | . . | . . | * | . . | * | . . |  |
| 6 | M | 70 | . . | † | * | . . | 8 | over 14½ | Less | . . | . . | * | . . | * | . . |  |
| Total 6 | 6 | Ave. 52 | 0 | 6 | 6 | 0 | Ave. 7 | Ave. 34 | Same 2<br>Less 4 | 2 | 2 | 2 | 2 | 4 | 0 |  |

## TABLE III.—FRACTURES OF LOWER END OF FEMUR

| No. | Sex. | Age. | Treatment. |  | X-ray. |  | Weeks in hospital. | Weeks from work. | Earning power. | Functional result. |  |  | Anatomical result. |  |  | Died. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Operative. | Non-operative. | Early. | Late. |  |  |  | Good. | Mod. | Bad. | Good. | Mod. | Bad. |  |
| 1 | M | 6 | . . | C | * | . . | 4 | 4 | Same | * | . . | . . | * | . . | . . |  |
| 2 | M | 30 | . . | E and C | * | . . | 7½ | 28 | Same | * | . . | . . | * | . . | . . |  |
| 3 | M | 35 | Plate | . . . . | . . | . . | 11 | 34 | Less | . . | * | . . | * | . . | . . |  |
| 4 | M | 38 | . . | E and C | * | . . | 2 | 28 | Less | . . | . . | * | . . | * | . . |  |
| 5 | M | 61 | . . | † | . . | . . | 3 | . . | . . . . | . . | . . | . . | . . | . . | . . | Delirium tremens |
| Total 5 | 5 | Ave. 34 | 1 | 4 | 3 | 0 | Ave. 5½ | Ave. 23½ | Same 2<br>Less 2 | 2 | 1 | 1 | 3 | 1 | 0 | 1 |

TABLE IV.—FRACTURES OF SHAFT OF FEMUR

| No. | Sex. | Age. | Treatment. | | X-ray. | | Weeks in hospital. | Weeks from work. | Earning power. | Functional result. | | | Anatomical result. | | | Died. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Operative. | Non-operative. | Early. | Late. | | | | Good. | Mod. | Bad. | Good. | Mod. | Bad. | |
| 1 | F | 3 | .. | E | * | .. | 7 | 13 | Same | * | .. | .. | * | .. | .. | |
| 2 | M | 3 | .. | VE | * | .. | 8 | 8 | Same | * | .. | .. | * | .. | .. | |
| 3 | M | 3 | .. | H and E | * | .. | 8 | 8 | Same | * | .. | .. | * | .. | .. | |
| 4 | M | 4 | .. | E and C | .. | .. | 9 | 12 | Same | * | .. | .. | * | .. | .. | |
| 5 | M | 4 | .. | E and C | .. | .. | 6 | 9 | Same | * | .. | .. | * | .. | .. | |
| 6 | M | 5 | .. | E | * | * | 9½ | 11 | Same | * | .. | .. | * | .. | .. | |
| 7 | M | 5 | .. | H and E | * | * | 8 | 9 | Same | * | .. | .. | * | .. | .. | |
| 8 | M | 6 | .. | H and E | * | .. | 12 | 17 | Same | * | .. | .. | * | .. | .. | |
| 9 | M | 6 | .. | H and E | * | .. | 6 | 8 | Same | * | .. | .. | * | .. | .. | |
| 10 | M | 6 | .. | C | * | .. | 7 | 9 | Same | * | .. | .. | * | .. | .. | |
| 11 | F | 7 | .. | E and C | * | * | 8 | 10 | Same | * | .. | .. | .. | .. | * | |
| 12 | M | 7 | .. | H and E | * | .. | 11½ | 11½ | Same | * | .. | .. | * | .. | .. | |
| 13 | M | 8 | .. | E and SB | .. | .. | 8½ | 13 | Same | * | .. | .. | * | .. | .. | |
| 14 | M | 10 | .. | E | .. | .. | 6 | 9 | Same | * | .. | .. | * | .. | .. | |
| 15 | M | 11 | Plate | .... | * | * | 11 | 14 | Same | * | .. | .. | * | .. | .. | |
| 16 | M | 13 | .. | E and C | * | .. | 6½ | 12 | Same | * | .. | .. | * | .. | .. | |
| 17 | M | 13 | .. | C | .. | .. | 7 | 14 | Same | * | .. | .. | .. | * | .. | |
| 18 | M | 14 | Plate | .... | * | * | 15 | 19 | Same | .. | * | .. | * | .. | .. | |
| 19 | M | 15 | .. | E | * | * | 12½ | 30 | Same | * | .. | .. | .. | .. | * | |
| 20 | M | 16 | Plate | .... | * | * | 12 | 20 | Same | * | .. | .. | .. | * | .. | |
| 21 | M | 18 | .. | Splint | * | * | 13 | 17 | Same | * | .. | .. | * | .. | .. | |
| 22 | M | 18 | .. | Splint | * | * | 13 | 17 | Same | * | .. | .. | * | .. | .. | |
| 23 | M | 21 | .. | † | .. | .. | 1 day | .. | .... | .. | .. | .. | .. | .. | .. | Shock |
| 24 | M | 23 | Plate | .... | * | * | 13 | 21 | Same | * | .. | .. | .. | * | .. | |
| 25 | M | 34 | .. | E and C | .. | .. | 7½ | 19 | Less | .. | * | .. | * | .. | .. | |
| 26 | M | 34 | Plate | .... | * | * | 10 | 26 | Same | * | .. | .. | * | .. | .. | |
| 27 | M | 38 | .. | Splint | * | * | 8 | 24 | Same | * | .. | .. | * | .. | .. | |
| 28 | M | 45 | .. | C | .. | .. | 9 | 20 | Same | * | .. | .. | .. | * | .. | |
| 29 | M | 47 | .. | E and C | * | * | 59 | 115 | Less | .. | .. | * | .. | .. | * | |
| 30 | M | 50 | .. | † | .. | .. | 3 days | .... | .. | .. | .. | .. | .. | .. | .. | Fractured skull |
| 31 | M | 50 | .. | C | .. | * | 6 | 12 | Same | * | .. | .. | * | .. | .. | |
| 32 | M | 50 | .. | E and SB | * | * | 6½ | 9½ | Same | * | .. | .. | * | .. | .. | |
| 33 | M | 52 | .. | HE and C | * | * | 7 | 21 | Same | .. | .. | * | .. | .. | * | |
| 34 | M | 53 | .. | E | .. | .. | 15 | 26 | Less | .. | * | .. | .. | * | .. | |
| 35 | M | 73 | .. | E and C | * | * | 14 | Idle | Same | .. | * | .. | .. | * | .. | |
| 36 | F | 74 | .. | † | .. | .. | 7 | Idle | Same | * | .. | .. | * | .. | .. | |
| Total 36 | M F<br>33 3 | Ave. 23⅓ | 5 | 31 | 24 | 17 | Ave. 10 | Ave. 16.9 | Same 31<br>Less 3 | 28 | 4 | 2 | 24 | 6 | 4 | 2 |

TABLE V.—FRACTURE OF FEMUR. FRACTURE LEVEL ACCORDING TO AGE WITH AVERAGE DISABILITY IN WEEKS

| | Total. | Under 15 years. | | | 15 to 45. | | | 45 to 60. | | | 60 and over. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Non-operative. | Opera-tive. | Average disability. | Non-operative. | Opera-tive. | Average disability. | Non-operative. | Opera-tive. | Average disability. | Non-operative. | Opera-tive. | Average disability. |
| Neck . . . . | 15 | 1 | . . | 10 | 1 | . . | 30 | . . | . . | . . . . | 12 | . . | 25 |
| Trochanters . | 6 | . . | . . | . . . . | 1 | . . | 27.5 | 4 | . . | 17.3 | 1 | . . | 14.5 |
| Shaft, Upper . | 5 | 2 | . . | . . . . | 2 | 1 | . . . . | . . | . . | . . . . | . . | . . | . . . . |
| Middle | 28 | 13 | 2 | . . . . | 4 | 1 | . . . . | 7 | . . | . . . . | 1 | . . | . . . . |
| Lower . | 1 | . . | . . | . . . . | . . | . . | . . . . | . . | . . | . . . . | 1 | . . | . . . . |
| ? . . | 2 | 1 | . . | . . . . | . . | 1 | . . . . | . . | . . | . . . . | . . | . . | . . . . |
| Total . . | (36) | (16) | (2) | 11.3 | (6) | (3) | 21.7 | (7) | . . | 34 | (2) | . . | Over 52 |
| Lower end . . | 5 | 1 | . . | 4 | 2 | 1 | 22 | . . | . . | 1 | . . | . . | * |
| Sum totals . . | 62 | 18 | 2 | . . . . | 10 | 4 | . . . . | 11 | 0 | . . . . | 15 | 0 | . . . . |

* The 11 cases which died early are not considered in Average Disability.
? Region of shaft not stated.

## TABLE VI.—Fracture of Femur. Disability Period According to Anatomical Result and Age

| Period of disability. | Total. | Anatomical result. | | | | Under 15 years. | 15–45 | 45–60 | Over 60 |
|---|---|---|---|---|---|---|---|---|---|
| | | Good. | Moderate. | Bad. | Died. | | | | |
| Under 6 weeks | 11 | 1 | .. | .. | 10 | 1 | 1 | 1 | 8 |
| 6 to 12 weeks | 16 | 16 | .. | .. | .. | 12 | .. | 4 | .. |
| 3 to 6 months | 19 | 11 | 5 | 2 | 1 | 7 | 7 | 2 | 3 |
| 6 to 9 months | 8 | 4 | 3 | 1 | .. | .. | 6 | 1 | 1 |
| 9 to 12 months | 1 | .. | 1 | .. | .. | .. | .. | 1 | .. |
| Over 12 months | 4 | 1 | 2 | 1 | .. | .. | .. | 2 | 2 |
| Not stated | 3 | .. | 3 | .. | .. | .. | .. | 1 | 2 |
| Total | 62 | 33 | 14 | 4 | 11 | 20 | 14 | 12 | 16 |

## TABLE VII.—Fracture of Femur. Functional and Anatomical Results According to Age (Non-operative and Operative)

| | Total. | Under 15. | | 15 to 45. | | 45 to 60. | | 60 and over. | |
|---|---|---|---|---|---|---|---|---|---|
| | | Non-operative. | Operative. | Non-operative. | Operative. | Non-operative. | Operative. | Non-operative. | Operative. |
| Functional: | | | | | | | | | |
| Good | 36 | 18 | 1 | 5 | 3 | 5 | .. | 4 | .. |
| Moderate | 9 | .. | 1 | 2 | 1 | 3 | .. | 2 | .. |
| Bad | 6 | .. | .. | 2 | .. | 3 | .. | 1 | .. |
| Death | 11 | .. | .. | 1 | .. | 1 | .. | 9 | .. |
| Anatomical: | | | | | | | | | |
| Good | 33 | 17 | 2 | 5 | 2 | 4 | .. | 3 | .. |
| Moderate | 14 | 1 | .. | 2 | 2 | 5 | .. | 4 | .. |
| Bad | 4 | .. | .. | 2 | .. | 2 | .. | .. | .. |

## TABLE VIII.—Fracture of Femur. Functional Results According to Anatomical Results

| | Total. | Functional. | | |
|---|---|---|---|---|
| | | Good. | Moderate. | Bad. |
| Anatomical: | | | | |
| Good | 33 | 30 | 3 | .. |
| Moderate | 14 | 5 | 6 | 3 |
| Bad | 4 | 1 | .. | 3 |
| Total | 51 | 36 | 9 | 6 |

## TABLE IX.—Fracture of Femur. Summary Table

| Location. | Number | Sex. | | Age in years. | | | Treatment. | | Time in hospital in weeks. | | | | Weeks absent from work. | | | | Earning capacity. | | Functional result. | | | Anatomical result. | | | Died. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Male. | Female. | Average. | Old. | Young. | Operative. | Non-operative. | Total. | Average. | Long. | Short. | Total. | Average. | Long. | Short. | Same. | Less. | Good. | Moderate. | Bad. | Good. | Moderate. | Bad. | |
| Neck | 15 | 4 | 11 | $64\frac{1}{2}$ | 84 | 14 | 0 | 15 | 100 | $6\frac{2}{3}$ | $29\frac{1}{2}$ | 2 | 90 | $22\frac{1}{2}$ | 30 | 10 | 7 | 8 | 4 | 2 | 1 | 3 | 3 | 1 | 8 |
| Trochanter | 6 | 6 | 0 | 52 | 88 | 46 | 0 | 6 | 41 | 7 | 10 | 4 | 204 | 34 | 91 | $9\frac{1}{2}$ | 2 | 4 | 2 | 2 | 2 | 2 | 4 | 0 | 0 |
| Shaft | 36 | 33 | 3 | $23\frac{1}{3}$ | 74 | 3 | 5 | 31 | 362 | 10 | 59 | $\frac{1}{7}$ | 571 | $16\frac{9}{10}$ | 115 | 8 | 31 | 5 | 29 | 4 | 1 | 25 | 6 | 4 | 2 |
| Lower extremity | 5 | 5 | 0 | 34 | 61 | 6 | 1 | 4 | $27\frac{1}{2}$ | $5\frac{1}{2}$ | 11 | 2 | 94 | $23\frac{1}{2}$ | 34 | 4 | 2 | 2 | 2 | 1 | 1 | 3 | 1 | 0 | 1 |
| Totals | 62 | 48 | 14 | 37 | 88 | 3 | 6 | 56 | 530 | 9 | 59 | $\frac{1}{7}$ | 959 | 20 | 115 | 4 | 42 | 19 | 37 | 9 | 5 | 33 | 14 | 5 | 11 |

Note:

| | Under 15 years. | Over 15 years. |
|---|---|---|
| Neck | 1 | 14 |
| Trochanters | 0 | 6 |
| Shaft | 19 | 17 |
| Lower end | 1 | 4 |

Result of Operated Cases

| | |
|---|---|
| Shaft | 4 good and 1 moderate functional.<br>4 good and 1 moderate anatomical. |
| Lower end | 1 good anatomical and moderate functional. |

# END-RESULTS IN FRACTURES OF BOTH BONES OF THE LEG[1]

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In presuming to bring forward the subject of fractures of both bones of the leg, this paper has no intention of discussing the question of treatment. It will limit itself to a statement of the end-results in a series of 40 cases, that being the number I was able to follow up of 288 cases treated in the Episcopal Hospital between the years 1905 and 1915.

Of the 40 cases followed, 6 were compound and 34 not compound fractures, *i. e.*, they were either simple or comminuted.

Group I classes cases as to age, sex, and leg involved, as follows:

| Years. | No. cases. | Male. | Female. | Right. | Left. |
|---|---|---|---|---|---|
| 0–14 | 7 | 6 | 1 | 3 | 3 |
| 15–44 | 22 | 13 | 9 | 13 | 8 |
| 45–59 | 9 | 7 | 2 | 5 | 4 |
| 60– | 2 | 0 | 2 | 2 | 0 |
| | — | — | — | Two cases unnoted | |
| Total | 40 | 26 | 14 | 23 | 15 |

As can be seen from the table, by far the greater number of cases occurred between the ages of fifteen and forty-four years, 22 out of 40, or 55 per cent. Males were involved in 65 per cent., and 57.5 per cent. of the fractures occurred in the right leg.

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[1] Read by invitation before the Philadelphia Academy of Surgery, May 3, 1915. Reprinted from Annals of Surgery, 1915, lxii, 619.

Group II gives us one of the most interesting classifications of the series. Here I have endeavored to show the effect of the anatomical result on the period of disability. The period of disability has been taken to mean the number of weeks the patient was kept from his work. The measurements were made from the lower border of the patella to the internal malleolus.

GROUP IIA (ALL CASES)

| Anatomical result. | Amt. of shortening. | No. cases. | Aver. period disability. |
|---|---|---|---|
| Good | 0–0.99 cm. | 17 | 14.8 |
| Moderate | 1.0–1.99 cm. | 16 | 21.7 |
| Bad | 2.0– cm. | 6 | 45.3 |

The above group includes all cases. The following table gives the same data for cases not compound:

GROUP IIB (CASES NOT COMPOUND)

| Anatomical result. | Amt. of shortening | No. cases. | Aver. period disability |
|---|---|---|---|
| Good | 0–0.99 cm. | 15 | 14.5 |
| Moderate | 1.0–1.99 cm. | 14 | 20.1 |
| Bad | 2.0– cm. | 4 | 42.0 |

While in the above tables, a shortening of from 1.0 to 1.99 cm. is classed as a moderate *anatomical* result, yet in practically all of these cases there was a very good *functional* result, the patients being able to do their former work without any loss of endurance or knowledge of the shortening of one leg.

Of all of the cases followed, there had been no open operative treatment in any of the simple or comminuted fractures. Two cases of comminuted fractures (*x*-ray photographs, Figs. 46, 47, 48, and 49) were treated by the Steinmann nail traction. Both of these cases healed with no shortening. The average period of disability for these 2 cases was 12.5 weeks, as compared with the average period of 15 weeks for the other 15 cases in the same group. Both cases were badly comminuted and, aside from the more rapid recovery and the perfect anatomical result, the Steinmann nail traction allowed

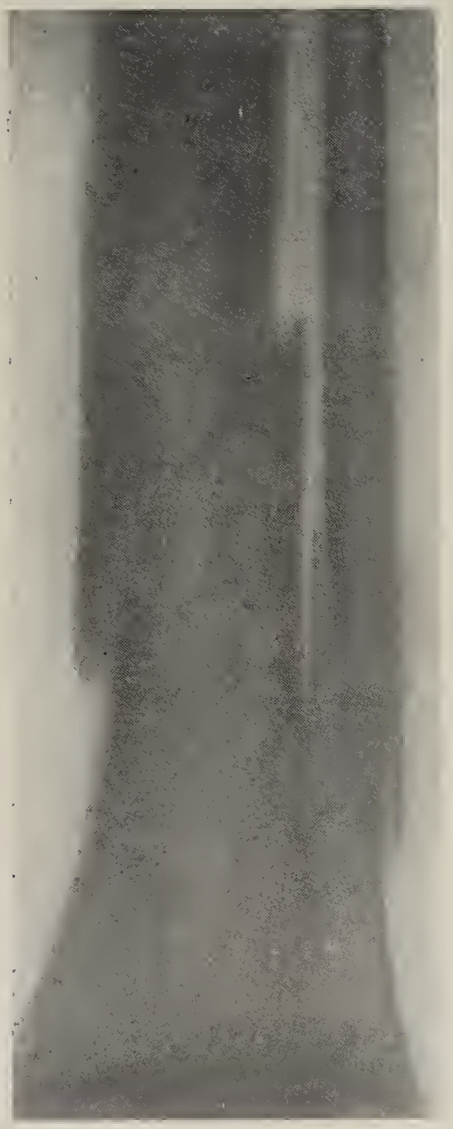

Fig. 46.—Comminuted fracture of tibia and fibula before treatment by Steinmann nail traction. (See Fig. 47.)

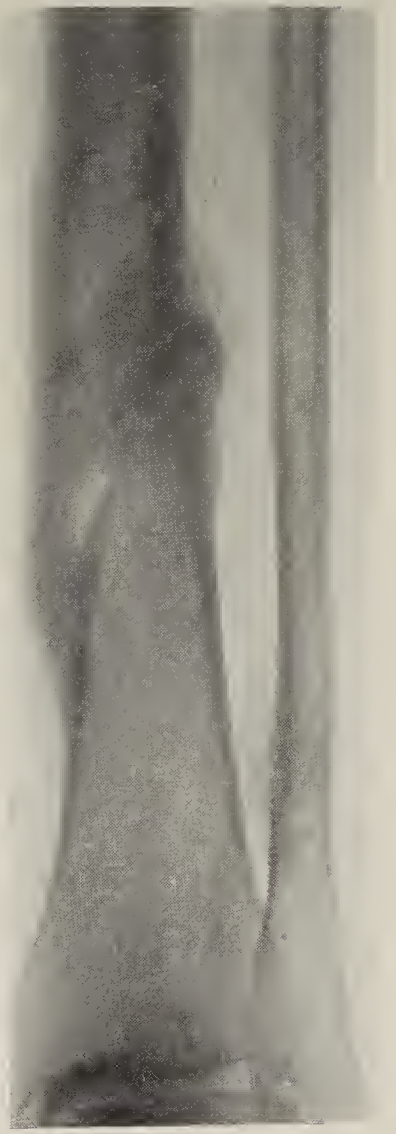

Fig. 47.—Comminuted fracture of tibia and fibula treated by Steinmann nail traction (sixteen pounds). Five months after injury; no shortening.

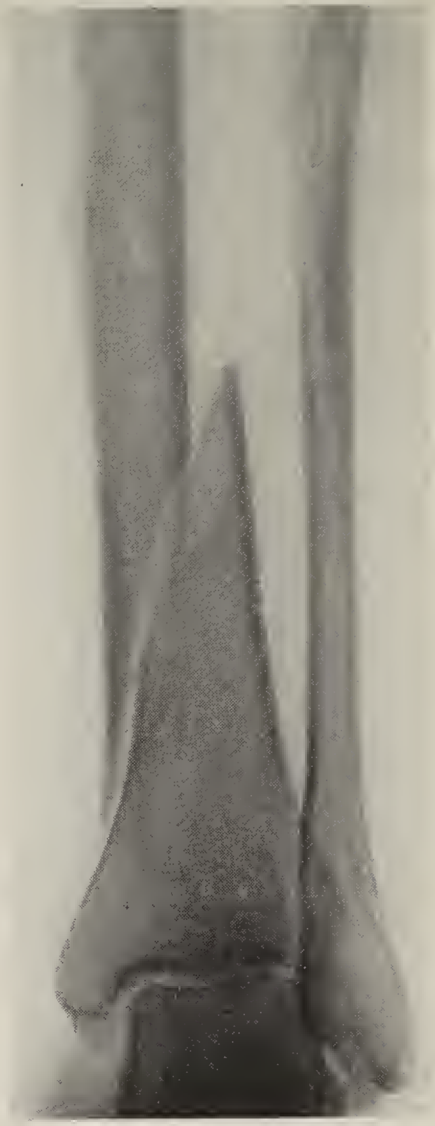

Fig. 48.—Comminuted fracture of tibia and fibula before treatment by Steinmann nail traction. (See Fig. 49.)

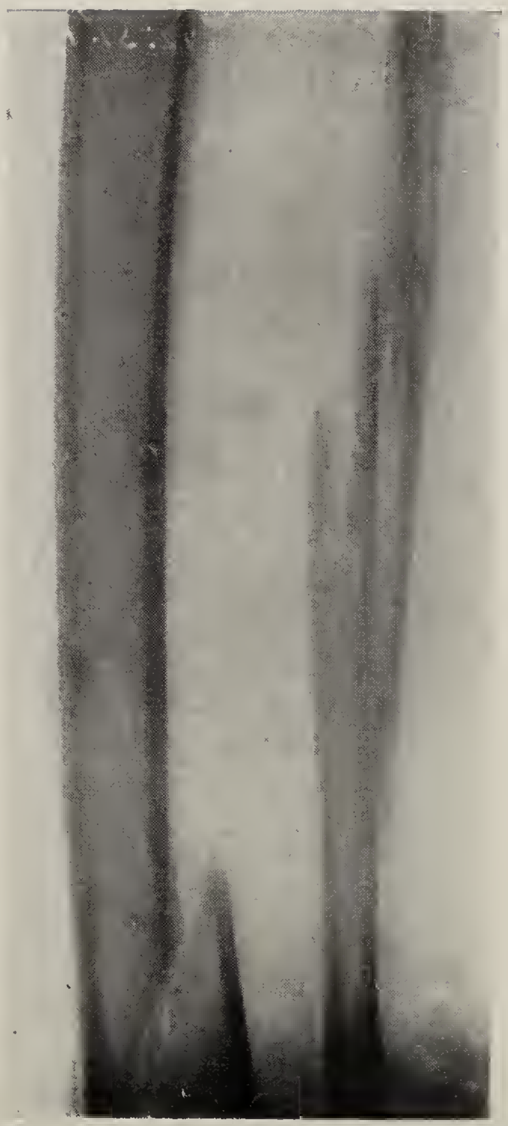

FIG. 49.—Comminuted fracture of tibia and fibula treated by Steinmann nail traction. Twenty days after injury; no shortening.

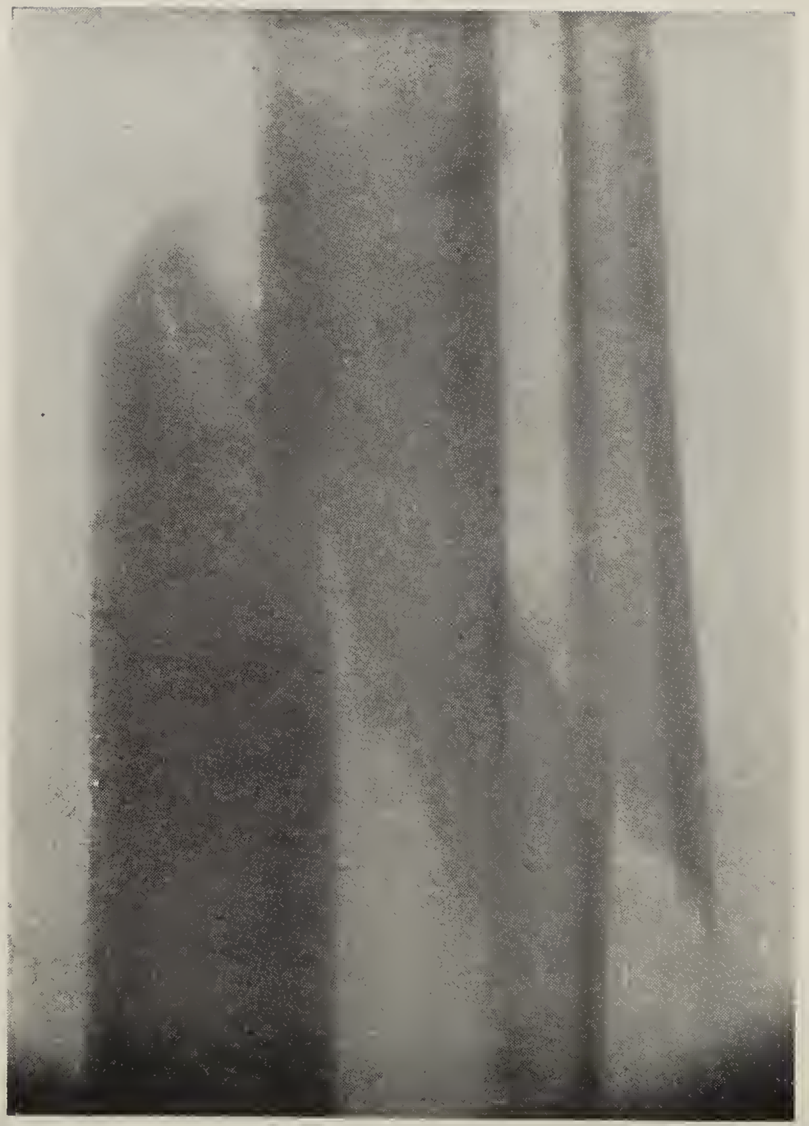

FIG. 50.—Compound fracture of tibia and fibula after Steinmann nail traction for twelve days (twenty-two pounds). (See Fig. 51.)

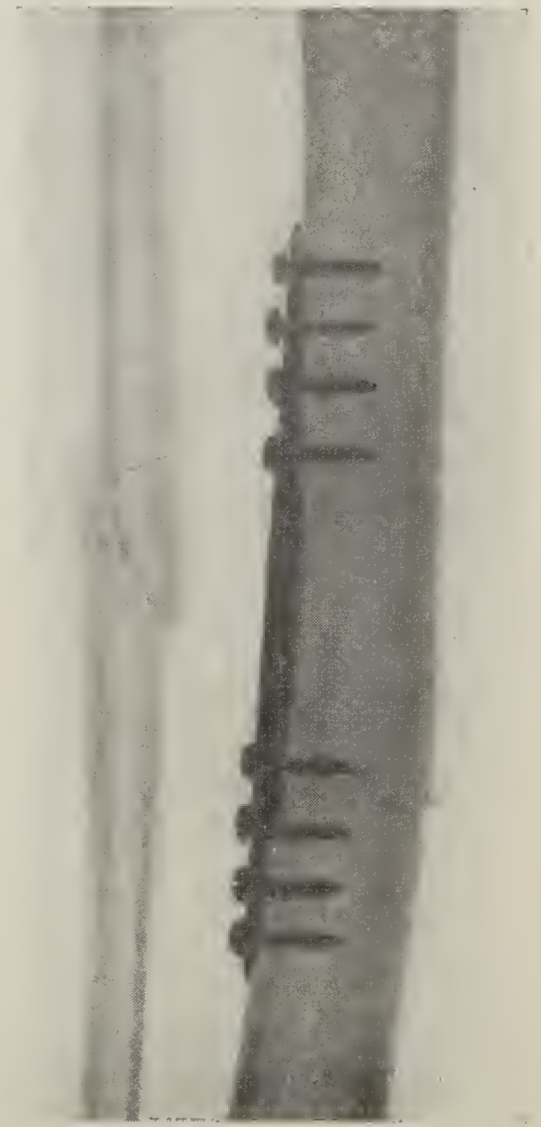

FIG. 51.—Compound fracture of tibia and fibula. Tibia plated eighteen days after injury; shortening, 1 cm.

these patients, while under constant traction, to move about a bit in bed, a fact which gave them considerable comfort and of which they speak with gratitude. That traction by means of the Steinmann nail is not always so satisfactory can be realized from a glance at Fig. 50. This case of compound fracture of both bones had a Steinmann nail driven through the os calcis on the sixth day after injury and twenty-two pounds of weight applied for twelve days, with no apparent reduction of the deformity. The bones were then exposed and the tibia plated, with an ultimately good anatomical and functional result (see Fig. 51).

The six compound cases of the series are grouped in the following table to show age periods and operative treatment:

GROUP III

| Years. | No. cases. | No oper. | Immed. oper. | Delayed oper. (after 10 days). |
|---|---|---|---|---|
| 0–14 | 2 | 1 | 1 | |
| 15–44 | 4 | .. | .. | 4 |
| 45–59 | 0 | | | |
| 60– | 0 | | | |

Group IV shows the compound cases grouped as to amount of shortening and average period of disability:

GROUP IV

| Amt. of shortening. | No. cases. | Aver. period of disability. |
|---|---|---|
| 0–0.99 cm. | 2 | 16 |
| 1.0–1.99 cm. | 2 | 64 |
| 2.0– cm. | 2 | 52 |

The following table gives the average period of disability in weeks for each region of bone considered, according to age periods:

GROUP V—AVERAGE PERIOD OF DISABILITY

| Years | Upper ⅓ | Middle ⅓ | Lower ⅓ |
|---|---|---|---|
| 0–14 | 11.5 (2) | 15 (2) | 15 (3) |
| 15–44 | 0 | 16 (2) | 23+(19) |
| 45–59 | 0 | 86 (2) | 21.4 (8) |
| 60– | 0 | 0 | 9 (2) |

The figures in parentheses represent the number of cases in each region. While Pott's fractures are not included in the series, it will be noted that the great majority of cases occurred in the lower third of the leg.

The conclusions to be drawn from these tables point to neither a strictly conservative nor a radical operative treatment as giving the best ultimate results. As so often stated, the majority of fractures occurred in males, between fifteen and forty years of age.

1. The right leg was affected in 57.5 per cent. of cases.
2. The average period of disability is directly dependent upon the anatomical result.
3. A moderate anatomical result may mean a perfect functional result.
4. Traction by means of the Steinmann nail would seem to be a hopeful compromise between a conservative and an operative treatment of obstinate cases.
5. The lower third of the bone is the most often involved but this fact has no apparent relation to age or period of disability.
6. The average time for reduction was on the day of injury except in 4 cases of compound fractures.

# UNUNITED FRACTURE OF THE NECK OF THE FEMUR, TREATED BY BONE-TRANSPLANT[1]

By ASTLEY P. C. ASHHURST, M.D.
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Dr. Astley P. C. Ashhurst presented a man, aged thirty years, who in August, 1913 (seven months after injury), came under his care at the Orthopædic Hospital, in Dr. Harte's service, and was found to have an ununited fracture of the neck of the right femur. He was unable to walk without crutches, on account of pain and weakness; he could stand alone, and even bear momentarily all his weight on the injured limb, but the hip grated, and the trochanter slid up and down on the pelvis. There was shortening of an inch and three-quarters. A skiagraph showed an ununited fracture at the base of the neck, oblique, and the longer fragment belonging to the head of the bone and the front of the neck (Fig. 52).

The patient was referred to the Episcopal Hospital (there being no vacant bed at the Orthopædic Hospital), and admitted to Dr. Frazier's service. Operation was done by Dr. Ashhurst on August 22, 1913.

1. An incision was made downward for 3½ inches from the anterior superior spine of the ilium, passing between the sartorius and tensor fasciæ femoris, and then between the ilio-psoas and rectus muscles. The capsule of the hip-joint was then opened and detached widely from the anterior intertrochanteric line, exposing the line of fracture, which was bevelled at the expense of the posterior surface of the neck, and extended anteriorly to the extracapsular region of the great trochanter. Only fibrous union was present, and the fragment were easily pried apart with a bone elevator. The fractured surfaces were then freshened. It was now found that by outward rotation, followed by longitudinal traction and finally by inward rotation, the fragments were jammed

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[1] Reprinted from Annals of Surgery, 1914, ii, 779.

together in good position. The wound was then temporarily packed with gauze.

2. A bone peg (Fig. 53) was removed from the crest of the left tibia by means of the speaker's circular saw (Fig. 54); the dimensions of this peg were four and a half inches long, and one-half by three-eighths by three-eighths of an inch thick (11.5 cm. long, and 1.5 cm. by 1 cm. by 1 cm. thick); it tapered slightly at its lower end. This bone peg was temporarily put in hot salt solution, and the leg wound closed.

3. The fracture of the cervix femoris was set under control of direct vision, by outward rotation, longitudinal traction, and finally inward rotation. While the limb was securely held in this position, an incision one and a half inches long was made over the great trochanter, and by means of a steel drill in a hand-driven brace (Fig. 54) a hole was bored through the trochanter and neck into the head. First a drill three-eighths of an inch in diameter was used, then one fifteen-thirty-seconds of an inch in diameter; and as the peg proved too large to be driven in through this hole, a drill half an inch in diameter was finally used. The peg fitted this hole very snugly, and it was necessary to drive it home with very vigorous blows from the mallet (Fig. 55). Some of the projecting end of the peg was then cut off. The two wounds in the hip were then closed, and the limb dressed in plaster of Paris from toes to axilla, in an abducted position. The time of the entire operation, including plaster of Paris, was two hours.

September 22, 1913. One month after operation, the plaster case was removed below the knee.

October 12, 1913. Seven weeks after operation the remainder of the plaster case was removed. There was no stiffness or thickening around the hip-joint, and the incisions were healed. The end of the bone peg was palpable beneath the skin over the great trochanter. There was no shortening of the limb and no movement between the fractured surfaces. Rotation at the hip was normal. Flexion to 150 degrees was easy and painless. The patient was to stay in bed two weeks longer.

October 20, 1913. Allowed to be up in wheel-chair.

October 25, 1913. Walking with crutches and a high shoe on the left foot, preventing any weight-bearing on the fractured limb.

November 27, 1913. Discharged from the ward, with direction to bear no weight on the right foot until six months after operation. A skiagraph made about this date showed the same conditions as Fig. 55. Figs. 56 and 57 are from photographs made December 1, 1913, four months and a half after operation.

Soon after this time the patient returned to his home in Ohio. About the first of the year (over four months after operation) he was permitted to abandon the use of the high shoe, but was directed to continue the use of crutches. This, however, he did not do. He felt so well and strong, he wrote, that as soon as he gave up using the high shoe he threw away his crutches. The effect of this was apparent when he next presented himself for observation on April 20, 1914. He was now wearing a lift in his right heel, as he felt the limb was shorter than the left. Measurements showed a shortening of half an inch; in December, 1913, there had been no shortening. Otherwise the hip was in good shape: there was flexion to about 70 degrees, free roation, abduction through about 10 degrees, and all these motions were painless. Walking was easy without any support, but there was a noticeable limp; the man had not been able to return to his usual occupation in the pottery, as this involved heavy lifting, and his employers were unwilling to risk an accident though the man himself said he was sure he could do the work. Meanwhile he had been doing odd jobs, but he had no steady employment. His chief complaint was of pain above the knee, worse after resting. When his knee gets limbered up he walks with very little limp.

A skiagraph was taken (Fig. 58), and this promptly revealed the cause of the shortening; the transplant had fractured, and the resulting coxa vara prevented as free abduction as had been present before the patient resumed weight-bearing on the limb. In spite of the fracture of the transplant, the fragments seemed to be firmly united, though little callus is visible in the skiagraph. It is interesting to see that the end of the bone peg projecting beyond the trochanter is gradually being absorbed. In Albee's similar case, of which an illustration is given in *Murphy's Clinics* (June, 1913, Fig. 98), it is stated that the skiagraph (five weeks after operation) shows proliferation of bone from the protruding end of the bone peg. In the skiagraph of the present patient there is apparent quite a growth of bone on the surface of the femur around the drill hole, but this, I believe, is due to extension from the femoral shaft and not due to growth in the transplant. Dr. Albee always preserves the periosteum in his transplants, and Dr. Murphy does likewise, but in Dr. Ashhurst's operations he has always removed the transplant subperiosteally, regarding the periosteum simply as a limiting membrane. McWilliams, however (*Jour. Am. Med. Assn.*, 1914, i, 346), has reported very careful experiments, in which he comes to the conclusion that it is very important to preserve the periosteum because it renders the transplant more easily permeable by surrounding capillaries, thus ensuring the early establishment of an adequate circulation through it. His experiments certainly show the value of preserving the periosteum, since in a number of instances he secured reproduction of bone from periosteum alone; but there may be another reason than that given by Dr. McWilliams for the greater liability of the bone graft to be absorbed when it is uncovered by periosteum. This may very well be that the periosteum really acts only as a limiting membrane, and *protects the transplant from solution by the cells of the surrounding tissues.* When the transplant is *embedded within osseous tissue,* as is the case in the transplant figured in the accompany illustrations and in the transplants used for splinting the tuberculous spine, there is apparently little fear of its absorption; when, on the other hand, it is embedded, not in osseous tissue but in the soft tissues, it is very likely to be absorbed unless protected from solution by an envelope of periosteum, its *normal limiting membrane.* Thus it is not surprising to see in Fig. 58 that the end of the bone peg projecting beyond the femur is being absorbed, while that embedded within bone, or in immediate contact with living bone, preserves its form unaltered. It will be interesting to know what became of the end of Albee's transplant eight months or more after operation. J. B. Murphy has reported one case (*Murphy's Clinics,* October, 1913, ii, p. 797) in which he thought the preservation of the periosteum was detrimental to the production of new bone, but unfortunately the stenographic report of his remarks is so inaccurate that it is impossible to know just what was done: the skiagraphs he presents indicate that a subperiosteal resection of the upper end of the humerus was done for cystic osteitis, and that in the place of the diseased humerus (removed subperiosteally) a transplant was inserted which had its own periosteum still in place. The subsequent skiagraphs (Figs. 200–204) indicate that the transplant with its periosteum was then surrounded by new bone formed between the transplant and the ensheathing periosteum which belonged to the excised humerus, and Dr. Murphy states that the periosteum which belonged to the transplant acted detrimentally in that even as long as seven months after operation it remained as a white line (visible in the skiagraph) between the transplant and the surrounding new-formed bone. But though all the skiagraphs clearly indicate that this new-formed surrounding bone developed beneath the original periosteum of the (subperiosteally)

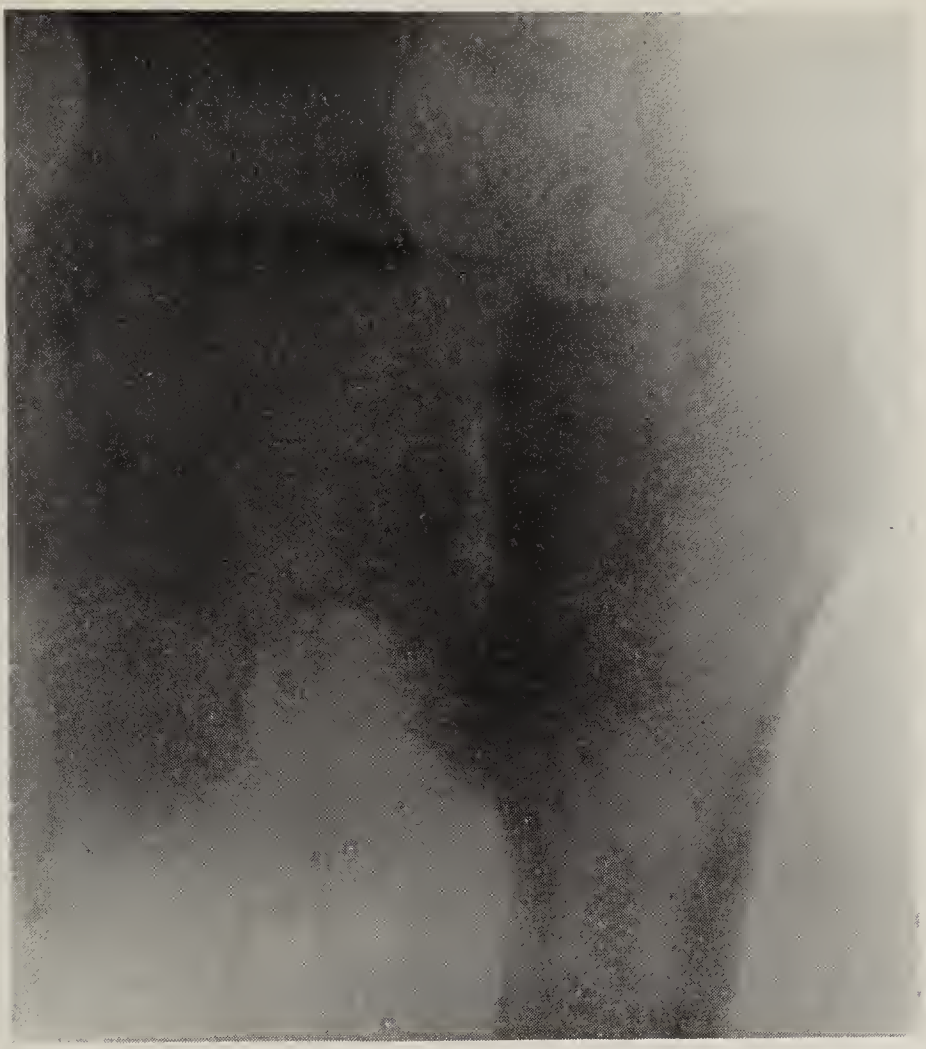


Fig. 52.—Ununited fracture of cervix femoris, seven months after injury. Unable to walk without crutches.

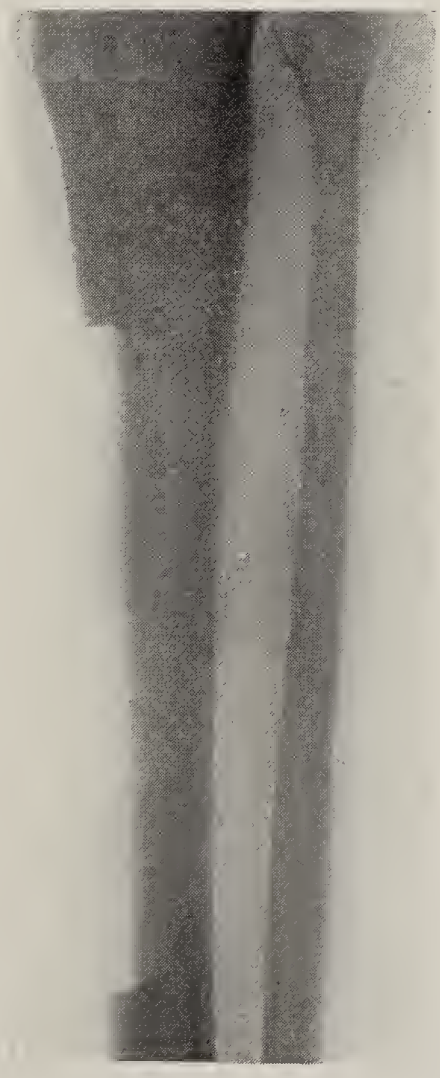


Fig. 53.—Showing defect in tibia after removal of bone peg.

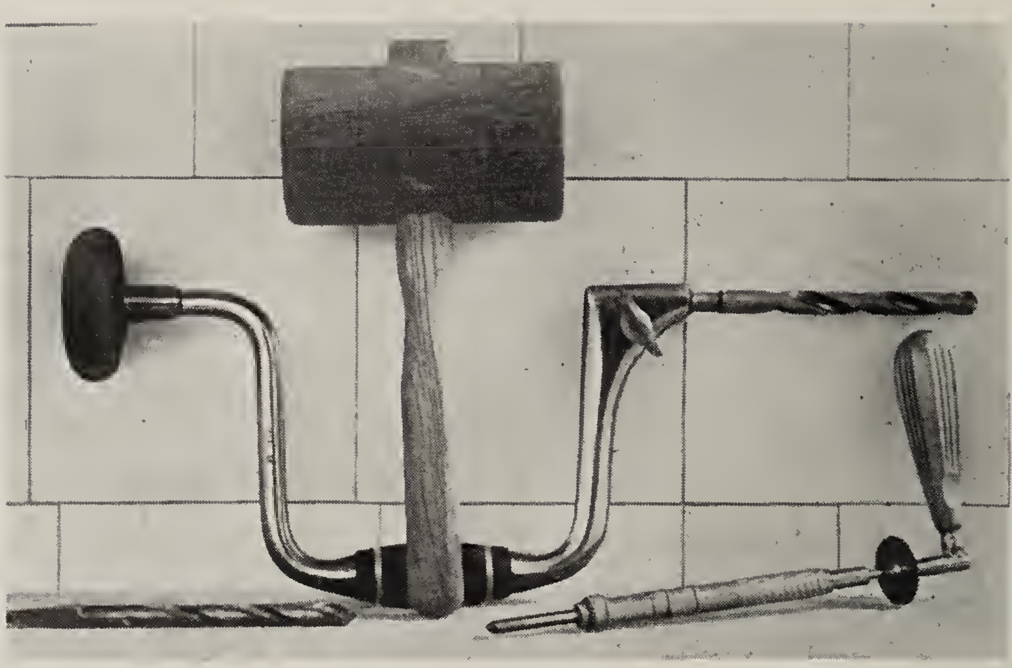
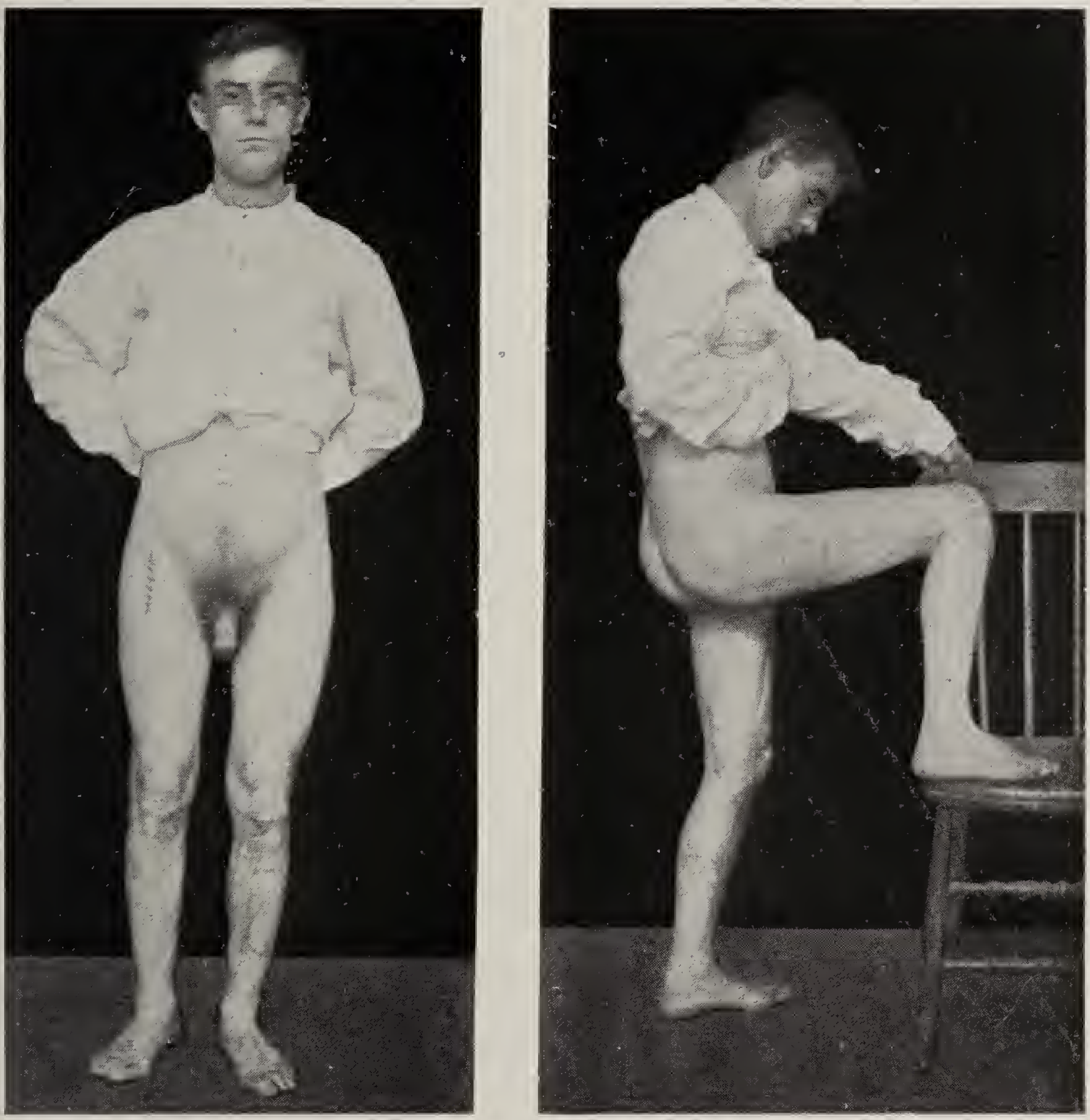


FIG. 54.—Circular saw, brace, drills and mallet used in transplanting a bone peg for ununited fracture of neck of femur.

FIG. 55.—Bone peg in ununited fracture of cervix femoris (skiagraph made through plaster-of-Paris dressing ten days after operation).



FIGS. 56 and 57.—Bone peg implanted in neck of femur for ununited fracture. No shortening, free motions, four and a half months after operation.

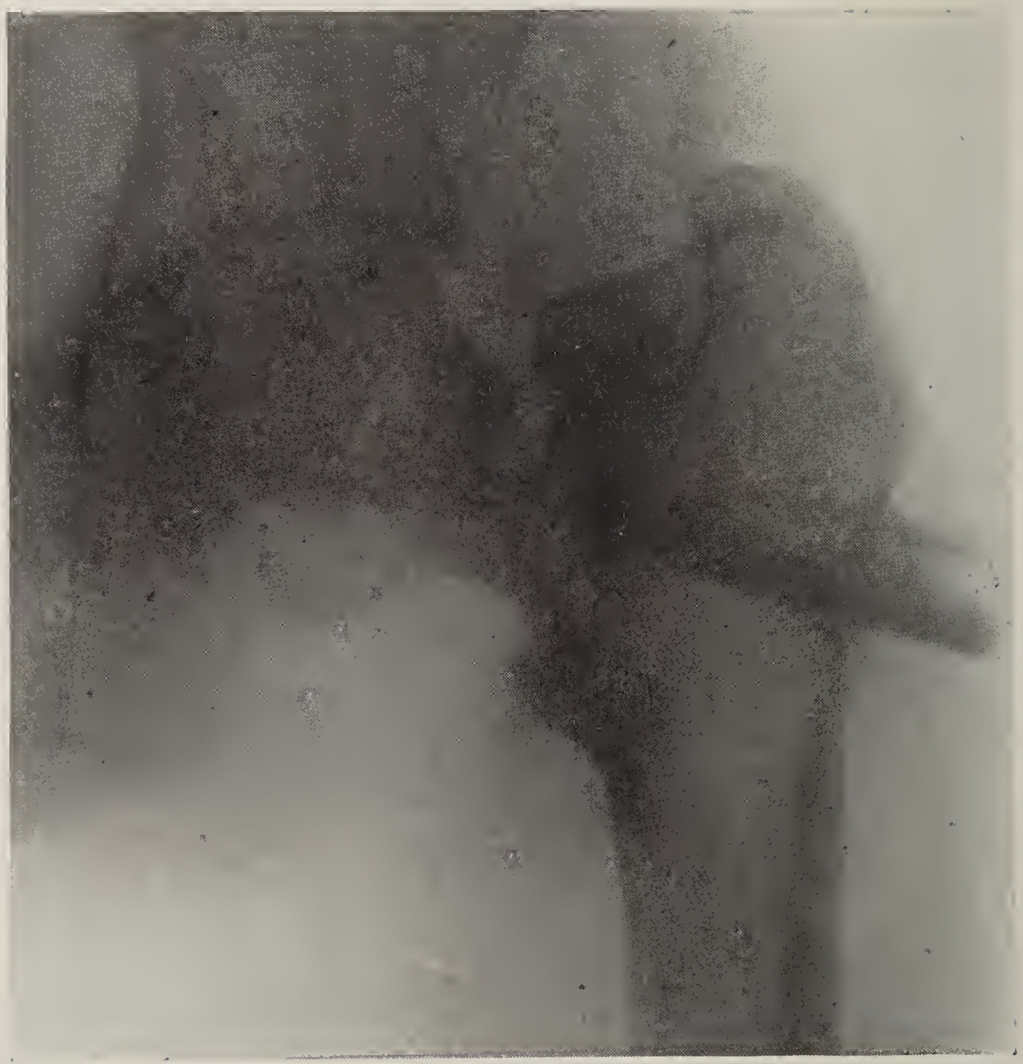


FIG. 58.—Skiagraph eight months after insertion of bone transplant, showing incomplete fracture of the transplant and secondary coxa vara.

excised humerus, it is stated in the text of *Murphy's Clinics* (p. 788) that the periosteum was excised along with the diseased humerus.

Although the patient presented has been walking on his leg without any support from crutch or cane for a period of four months, it is scarcely possible to reckon his present condition as an end-result. He has not returned yet to his ordinary work, but there seems every likelihood of his being able to do so within a short time.

Finally, Dr. Ashhurst suggested that bone transplantation is a better method of treating ununited fractures of the hip than the use of nails, screws, etc. But, as the present case demonstrated, it is not safe for the patient to bear weight on the limb as soon as four and a half months after operation. This patient did so contrary to advice, and as a consequence he fractured the transplant.

In an interesting paper on the subject of intracapsular fractures of the hip, read before this Academy two years ago by Dr. G. G. Davis (*Tr. Phila. Acad. of Surg.*, 1913, xv, 112), the following were among the conclusions reached:

"The surest way of remedying cases of ununited fracture of the neck of the femur is to cut down, freshen the edges of the fragments, pin them together with screws, nails, or other means, and put them up in the abducted position. When foreign bodies are inserted to pin the fragments together they are likely to cause discomfort sufficient to necessitate their removal. Considerable discomfort follows the operation and the patient is inclined to attribute this to the nail or screw and demand its removal. Firm union can be obtained by freshening the surfaces of the fragments and then jamming them together by widely abducting the limb and fixing it in plaster of Paris without the use of any nails, screws, or other fixation appliances."

These conclusions of Dr. Davis, who has had more experience than any other Fellow of the Academy with operations for ununited fractures of the hip, were reached before the operation of bone transplantation came into general use; and it seems that this is a better method of fixation than the use of foreign material such as nails, screws, or even ivory pegs; and that some form of direct fixation of the fragments is extremely desirable, though it had been shown by Dr. Davis that is some cases it was possible to secure firm bony union without direct fixation.

# RECENT TRAUMATIC DISLOCATIONS OF THE HIP WITH A CONSIDERATION OF THE RESULTS OF SEVEN CASES[1]

By CARL R. STEINKE, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

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Traumatic dislocation of the hip, according to various authors, comprises between 2 and 10 per cent. of all dislocations. That it is much more common in men than in women is well known.

First let us consider the types of dislocation of the hip. The two primary types depend upon whether the head of the femur is forced out of the acetabulum in front of (anterior type), or back of (posterior type) the line between the anterior superior spine of the ilium, and the tuberosity of the ischium, which is the summit of the two planes forming the external surface of the innominate bone. The posterior variety is subdivided into the high (iliac), middle (sciatic), and low (ischiatic), while the anterior may be high (pubic), middle (thyroid or obturator) or low (perineal). The posterior type, with its subdivisions, is by far the more frequent in occurence. That they are not at first always posterior but anterior and are brought into the posterior position, either by the patient endeavoring to use the limb, or by someone assisting the patient by attempting to turn in the everted foot or otherwise straighten the limb, is sometimes true. Thus it is difficult to determine the exact proportion of the posterior to the anterior type at the time the luxation is produced. The usual

[1] Reprinted from Annals of Surgery, 1914, ii, 617.

characteristics of flexion, adduction, and inversion are about the same in each of the posterior varieties; while flexion, abduction, and eversion make up the deformity in the anterior type. A rare type is a posterior dislocation with eversion.

The anterior type is generally produced by flexion and hyperabduction, followed by external rotation, making the thyroid type; a shift in the position of the thigh may then make it into a pubic or perineal position, or by further internal rotation a posterior variety may be produced. Luxation may be caused by flexion and strong inward rotation, together with hyperabduction, usually producing the posterior type.

The diagnosis, as a rule, is not difficult, as the deformity is characteristic. However, fracture of the neck of the femur must always be thought of and eliminated as a possibility either as a separate condition or in conjunction with a dislocation.

During the past nine years (up to January 1, 1914), 15 cases of dislocation of the hip have been treated in the Episcopal Hospital. Six of these were congenital in type, one pathological and one old traumatic case, leaving 7 cases to be considered in this article.

Case I.—M. M., Male. Admitted February 21, 1907 (file No. 619). The patient was admitted, giving a history of having had a large amount of coal fall upon him. On examination a high posterior dislocation of the left hip was found. The leg was adducted, inverted, and flexed. Pain, tenderness, and rigidity were noted about the hip. The patient was placed on a table face down. The knee and thigh were flexed and then direct traction made downward while an assistant guided the head of the femur by pressure on the great trochanter, thus reducing the luxation. Five days later the patient was discharged in good condition. Diagnosis: High posterior luxation of left hip.

At the time this article was prepared no trace of the patient could be found.

Case II.—J. M., male, aged fifty-five years. Admitted April 19, 1910 (file No. 1094). The patient was admitted with a lacerated scalp and shin and posterior dislocation of the right hip. The right thigh was flexed on the abdomen and adducted, while the knee was flexed and the foot turned in. He was unable to extend the thigh either voluntarily or by force, but could flex it on the abdomen. The great trochanter was felt anterior to its normal position and not prominent. The head of the femur could be felt in the sciatic area and could be felt to rotate with the thigh.

*Ether Anesthesia.* The leg was flexed on the thigh and thigh flexed on the abdomen in adduction, then circumducted outward and extended. The head of the femur was felt and heard to snap back into its normal position. The leg was placed in normal position between sand-bags.

April 22. Patient very sore and stiff over entire body especially right hip region.

April 25. Still tender about right hip, otherwise in good condition.

May 25. Up and around. Hip slightly painful but he walks on crutches.

June 4. Discharged in good condition. Diagnosis: Posterior (sciatic) luxation of right hip. No trace of patient could be made after leaving the hospital.

CASE III.—B. S., female, aged thirty-eight years. Admitted February 9, 1911 (file No. 473). One week previous, while attempting to cross railroad tracks, she was struck by the tender of the engine, the engine and fire-box passing over her, causing severe bruises and fractures. On admission she was found to have a fracture through the middle third of the right clavicle and a dislocated left hip. The left foot was inverted and rested on the instep of the opposite foot. The head of the femur (left) was palpated between the acetabulum and crest of the ilium.

February 10. Gas and ether anesthesia. Allis' method of reduction used. Head in good position. Buck's extension, with ten pounds applied.

March 3. Left hip-joint not particularily painful. Patient out of bed and there is no tendency to recurrence of the luxation. The right clavicle shows good union with slight deformity. Patient discharged in care of family physician. Diagnosis: Posterior (iliac) luxation of left hip.

In a letter from her physician of January 24, 1914, three years after the accident, he says concerning her hip condition: "She has disability of the left leg due to the dislocated hip, fractured pelvis, and numbness and loss of power due to nerve injury from deep laceration of the thigh." It would seem her present condition is probably not due so much to the dislocation as to the nerve injury received at the time of the accident.

Case IV.—H. K., male, aged twenty-eight years. Admitted June 7, 1912 (file No. 1834). Patient fell from a ten-foot fence and was brought to the hospital right away. The left leg was all right. The right thigh was flexed and fixed in internal rotation, while the foot lay in inversion. The head of the femur was felt on the flaring wing of the ilium. There was great pain on moving the leg. Some laxity of the iliotibial band.

Under chloroform anesthesia the luxation was reduced by flexion, traction, external circumduction and extension. The right leg then measurered one-quarter inch longer than the left.

June 8. Slight aching pain in affected hip. Able to invert and evert the toes fairly well.

June 15. General motion fairly free.

June 16. Up on crutches.

June 19. Discharged walking on crutches. No limitation of motion and no shortening. Diagnosis: Posterior (iliac) luxation of right hip. X-ray showed reduction of dislocation.

Case V.—B. L. S., male, aged sixty-one years. Admitted January 23, 1913, having jumped from a third story window to the ground, causing pain and disability of right hip. The right thigh, well in the position of flexion and internal rotation, cannot be brought into full extension. Measuring from the anterior superior spines to the internal malleoli gave one and one-quarter inches shortening of the right leg. The base of Bryant's triangle on the right was one-half inch shorter than the left. With the right thigh flexed the trochanter was felt one and one-half inches above Nélaton's line. Area of tenderness on level with anterior superior spine and about five inches from it on the right. Reduction by Stimson method (as in Case I) attempted for eleven minutes. Deformity improved but luxation not reduced.

January 24. X-ray shows luxation of hip. Patient placed in supine position on hard mattress on the floor. (Ether.) The thigh flexed on the pelvis to a right angle and leg at right angles to the thigh. With traction upward, internal and external rotation of the thigh, together with pressure on the trochanter the head of the femur was brought into position in the acetabulum. Buck's extension was applied with light weights.

February 10. General condition good.

February 17. Weights and straps removed. Much more comfortable.

February 24. Out of bed for a short time. No deformity of leg.

March 5. Up all day. General condition all right.

March 11. Discharged in good condition as cured. Diagnosis: Posterior luxation of right hip.

In a letter from the patient just a year after the accident, January 22, 1914, he says: "I run, hunt, climb ladders, skate and am able to work in the fields of my farm. I am a much better man physically than I was a year ago." This is sufficient evidence that he suffers no ill effects from the dislocation.

Case VI.—W. B., male, aged twenty-six years. Admitted May 8, 1913, with a history of having had a cart load of coal dump and strike him in the middle of the thigh, covering him with coal. He was unable to help himself out and had to be lifted onto a stretcher. He complained of pain in the right hip-joint and inability to walk. Examination: The head of the right femur palpated posterior and high up above the acetabular rim. The right leg was flexed, inverted and shortened. Ether was given and the dislocation reduced by the Allis method.

May 10. Condition good.

May 15. Discharged in good condition as cured. Diagnosis high (iliac) posterior dislocation of right femur.

A letter from the patient dated January 17, 1914, about eight months after the accident states as follows: "I went to work in three weeks' time. My hip at present is in good condition. I have no pain at all, and can go up and down stairs the same as ever." This case shows another good end-result.

Case VII.—F. McL., male, aged thirty-five years. Admitted December 8, 1913 (file No. 4974). While working on a roof a heavy

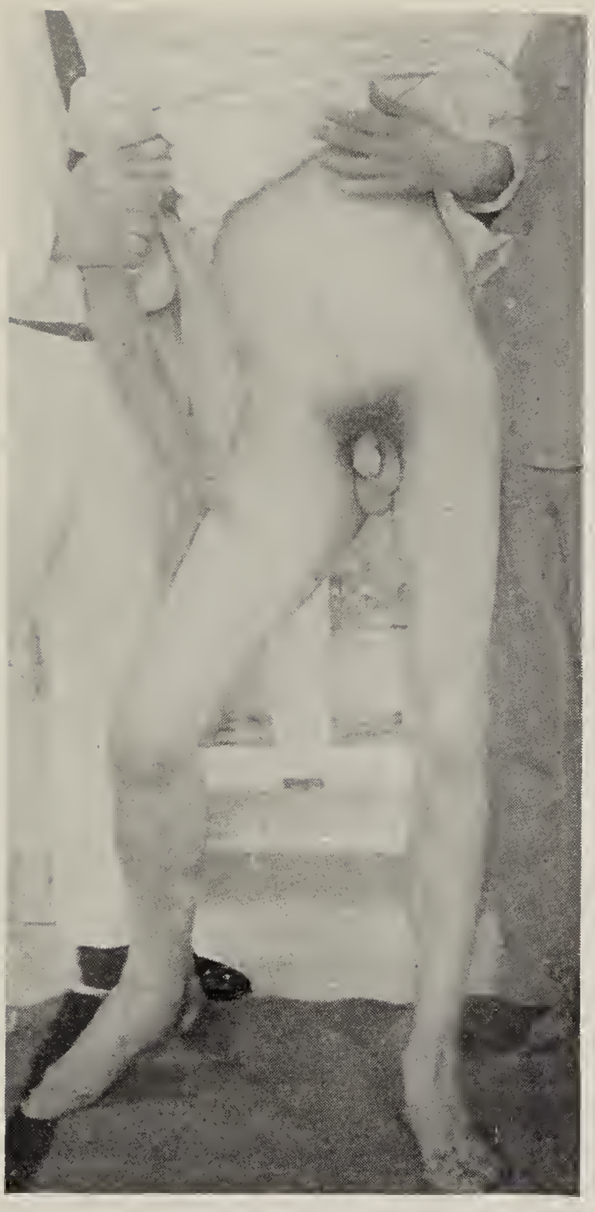

FIG. 59.—Case VII. Showing anterior luxation of right hip.

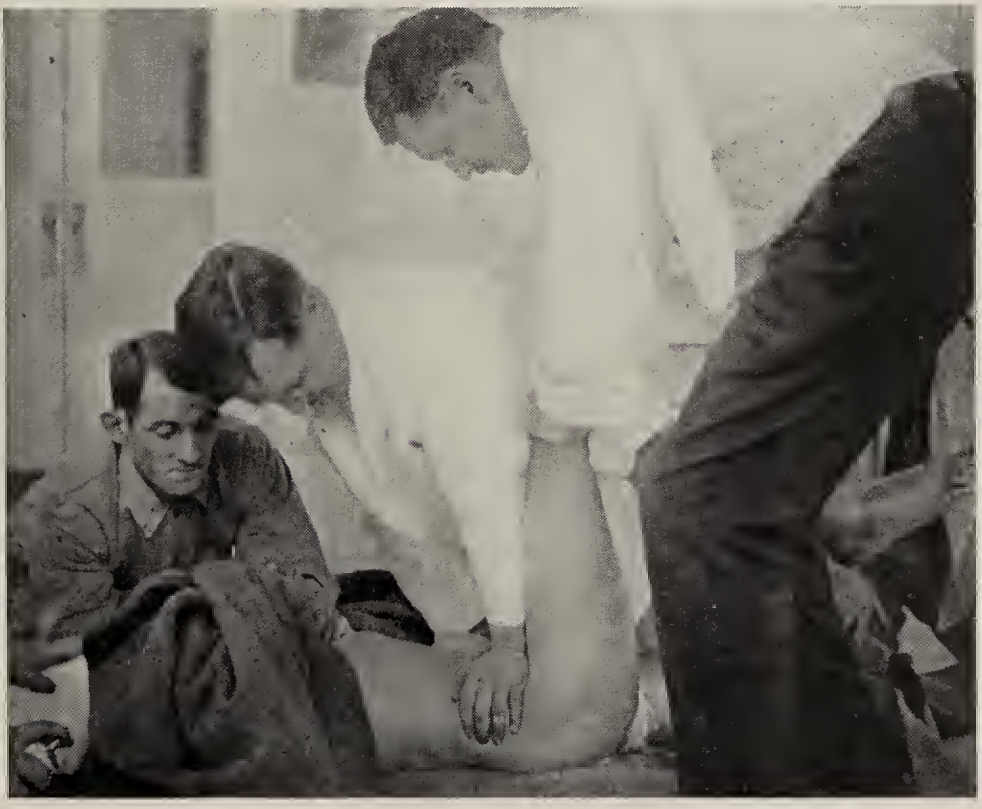

FIG. 60.—Showing method of reduction (direct) in Case VII.

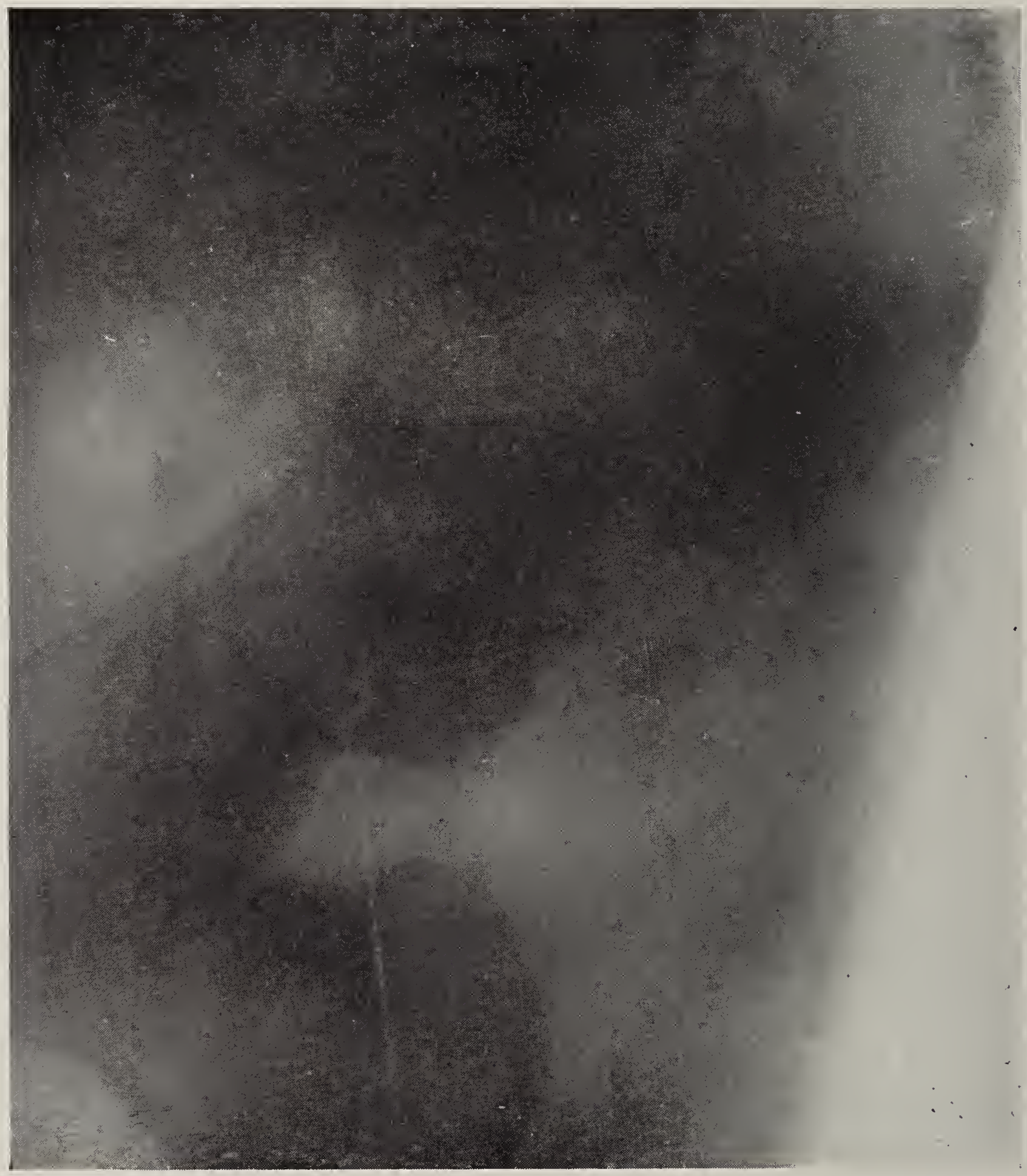

FIG. 61.—Case VII. X-ray No. 8722. Showing anterior dislocation of femur (right).

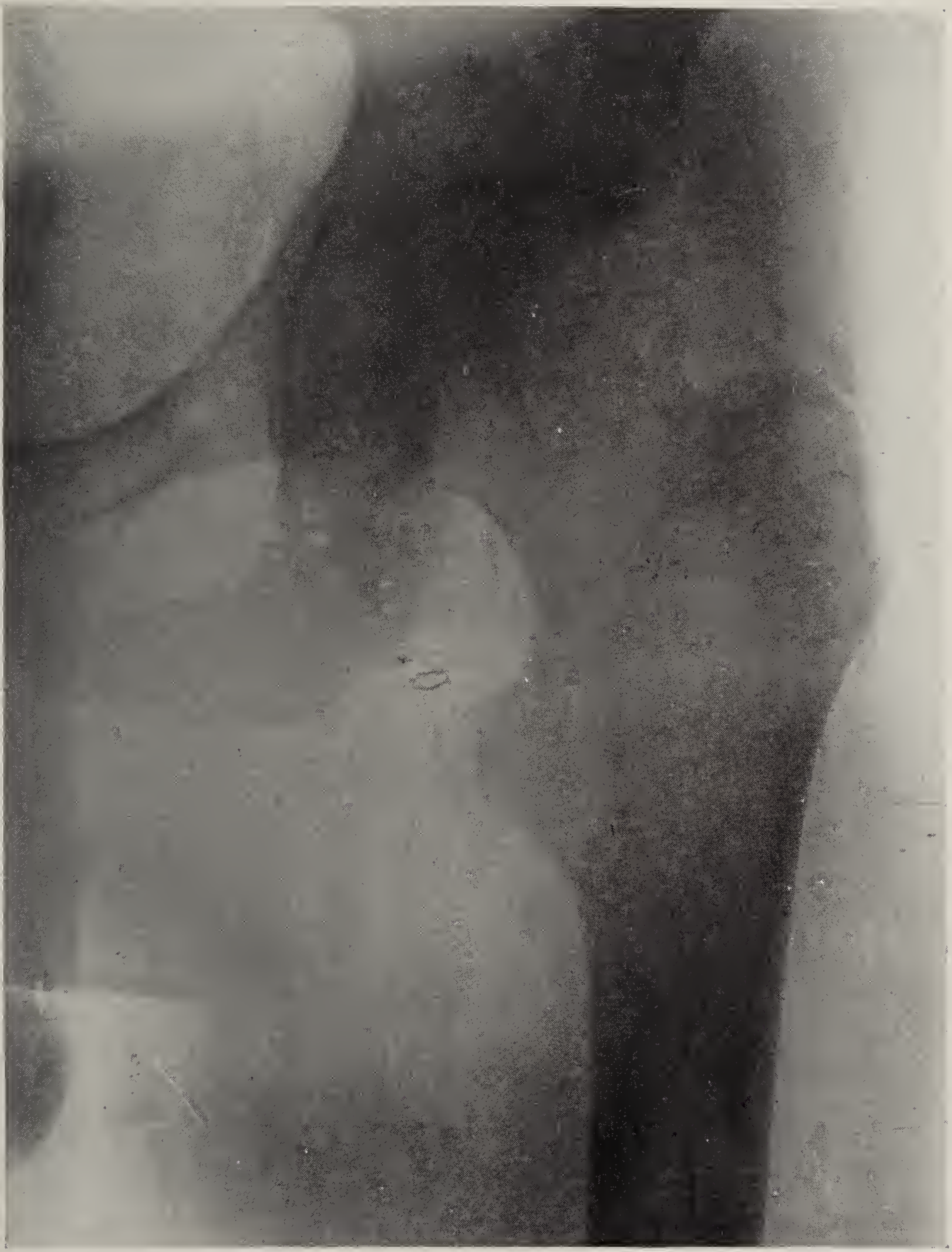

FIG. 62.—Case VII. X-ray No. 257*a*. Showing femoral head in position after reduction.

gust of wind struck him causing him to fall fifteen feet. He was brought to the hospital and examination revealed the following: Left leg normal. The right leg was flexed on the thigh and thigh on the abdomen. The leg could not be straightened. There was severe pain on motion. Leg was slightly abducted but no eversion of the foot, probably due to the position of the pelvis. On movement the head of the femur seemed to be dislocated but the exact position could not be located due to the rigidity of the adductors. There was apparent lengthening of the leg. On the posterior surface of the thigh was a large area of ecchymosis. There was also marked swelling of the thigh. With the patient standing on the left leg the right was found to be in the position of abduction with the thigh flexed, knee flexed and the toes resting on the floor in eversion (Fig. 59).

December 9. X-ray showed anterior luxation of right hip (Fig. 61).

December 13. Ether anesthesia. The patient was placed on his back upon a hard mattress. An assistant held the pelvis by downward pressure on the anterior superior spines. The right leg was flexed to a right angle on the thigh and the thigh at right angles to the body. By strong traction skyward, together with manipulation of the leg producing internal and external rotation of the thigh and at the same time having pressure made on the great trochanter, the femoral head was returned to its normal position with a snap that could be both heard and felt. Patient was then put to bed with sand-bags one to either side of the affected limb.

December 15. Still some pain in the right hip. Leg in good position.

December 24. Sand-bags removed and the patient allowed to get up on crutches.

December 26. Discharged in good condition, on the crutches. Diagnosis: Anterior (thyroid) dislocation of the right hip.

When seen seven weeks after the injury he said he had no pain in the hip and was walking about the house without even a cane. He had no disability but felt some fear of overdoing the leg.

In the above series of cases there are 6 males and 1 female. The age ranges from twenty-six to sixty-one years. The time in the hospital ranged from 5 to 47 days.

There is only one case of anterior luxation. Of the 6 posterior cases, 4 are stated as being high or iliac, 1 sciatic, while the sixth is simply given as posterior. This series confirms the statistical records as to the greater frequency of the posterior type.

The mode of reduction: Allis's method was used in 2 cases. Two others were reduced by flexion, with adduction, outward rotation and extension which from the description was the Allis method of retracing the steps gone through in the production of the luxation. One case was reduced with the patient in the prone position (Stimson method). The Stimson method failed in one of the cases. The only anterior and one of the posterior luxations were reduced by flexion of the thigh to a right angle with the body in the supine position and the leg at right angles to the thigh. Then by traction skyward, with internal and external rotation of the thigh together with pressure on the great trochanter the head of the femur was returned to the acetabulum. Thus it is noted that each of the methods was efficient in one or more cases.

The mode of treatment following the reduction varied. Three cases were simply kept in bed. Two had Buck's extension applied for a time following the reduction. The remaining 2 were kept in bed with a sand-bag to either side of the affected leg.

Only 4 cases could be traced at the time of preparing this paper. Three report themselves in the best of health and condition with no disability from the accident. The fourth is reported as having numbness and loss of power of the leg, due not only to dislocation of the hip, but to fracture of the pelvis and nerve injury as well. It seems fair to conclude from the above cases that simple luxation of the hip, when properly reduced, should not impair the function in the slightest degree.

# CASES ILLUSTRATING MODERN BONE AND JOINT SURGERY[1]

By ASTLEY P. C. ASHHURST, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

Even so short a time as twenty years ago bone and joint surgery consisted essentially in operations for necrosis, and excisions of joints for advanced tuberculous disease or to remedy disability due to ankylosis in bad position. Osteotomies were done for the deformities due to rachitis and for deformity at the hip-joint following healed coxalgia; and even in cases of marked disability from non-union of fractures operative fixation was undertaken, and in many cases with a great measure of success. An occasional bone cyst was opened and its interior scraped, but in most cases of tumors in bone amputation was done. Though modern methods have been gradually developed during this period it is only within the last five years that what may be described as modern bone, and joint surgery has begun to exist. The old operations are still done, but there has been added a large number of new procedures of utmost importance. These have been made possible largely by the perfection of aseptic technique, and may be discussed under the headings of *Treatment of Fractures*, *Treatment of Ankylosis*, and *Bone Transplantation*.

Treatment of Fractures. The general employment of the *x*-ray to control the reduction of bone fragments, and the increased interest of accident insurance companies in the results of treatment, have brought the treatment of fractures once again into a very important place in surgery. Indeed it is becoming almost a surgical specialty, and certainly it is true that most of the bad results are directly due to ignorance and carelessness in treatment. Patients with broken bones should be treated by surgeons whenever the latter are available. Though the general practitioner cannot always avoid having fracture cases under his care, he will do much better not to attempt to care for them if he can possibly avoid it; he has neither the training nor the facilities which are required to secure the best results.

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[1] Read before the College of Physicians of Philadelphia, October 7, 1914.

It may be admitted that accurate anatomical reduction of a fracture in the shaft of a long bone is not always necessary to secure perfect functional recovery, yet it is very desirable to secure such reduction, especially in the lower extremity, where shortening or axial displacement is much less easily compensated for than in the arm or forearm, and if the fracture is near a joint it is imperative to secure anatomical replacement of the fragments at the risk of lasting disability if this is neglected. Fortunately it often is possible to secure reduction and to maintain it without resort to formal operation, as the following instance shows:

*Fracture of Surgical Neck of Left Humerus Impacted in Bad Position; Reduction under Anesthetic.*

Case I.—Robert E., aged thirty-five years, was admitted to Dr. Frazier's service in the Episcopal Hospital, September 8, 1914. He had fallen thirty feet, and sustained, in addition to the injury mentioned above, fractures of the left radius and ulna above the wrist, and a fracture of the left femur below the trochanters. Here was a case of multiple fractures, in a young and healthy adult. Fig. 63 shows the position of the shoulder fragments on admission: the shaft was impacted into the head, but the position was very bad indeed, and reduction was imperative. The day after admission the patient was etherized, and the impaction at the shoulder was broken up by manipulation, the fractured ends reduced, and a suitable dressing applied. Fig. 64 is from a skiagraph made after reduction. This shows that the upper fragment is rotated slightly outward; to meet this indication the arm was

FIG. 63.—Case I, impacted fracture of surgical neck of humerus.

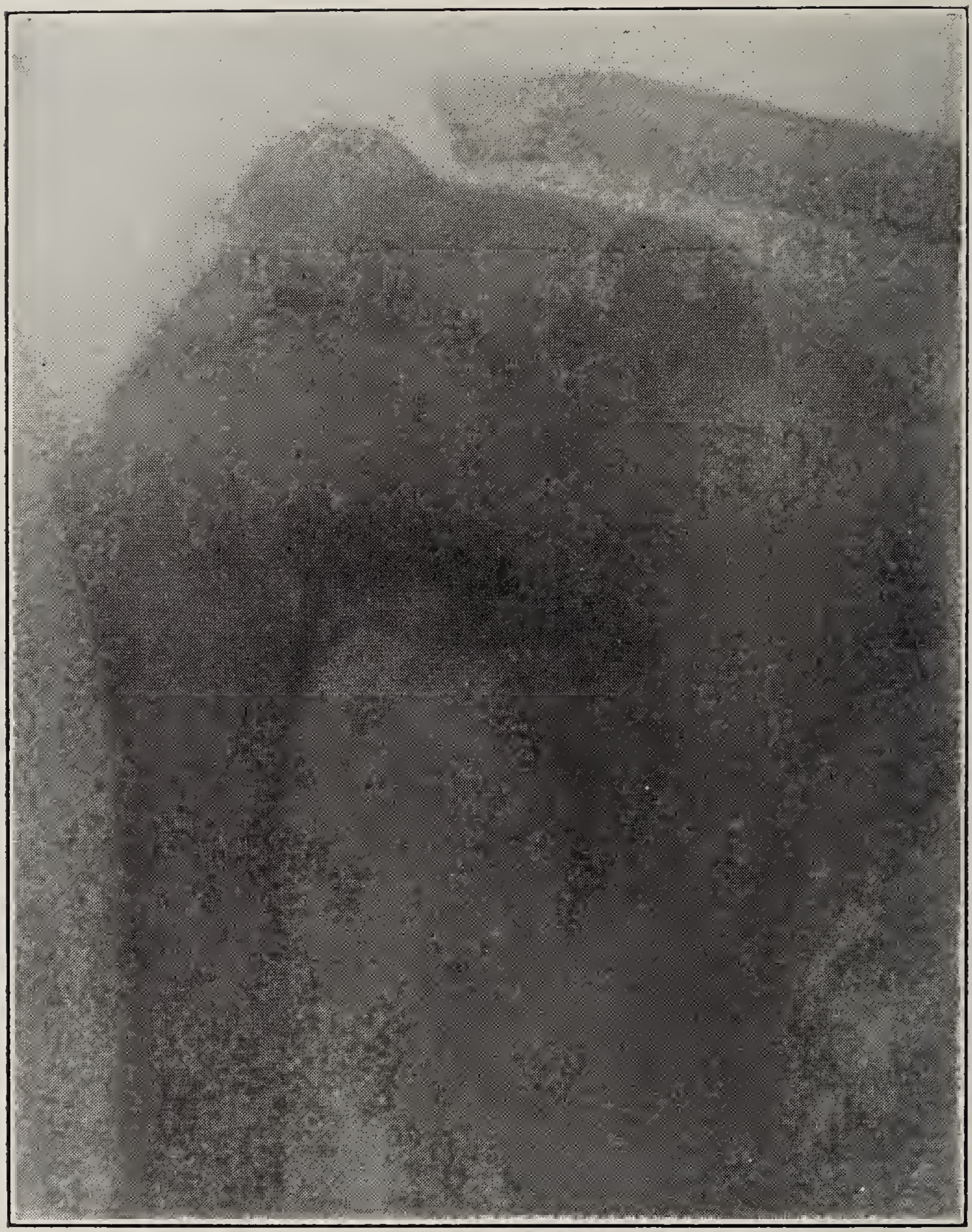

Fig. 64.—Case I, condition after reduction.

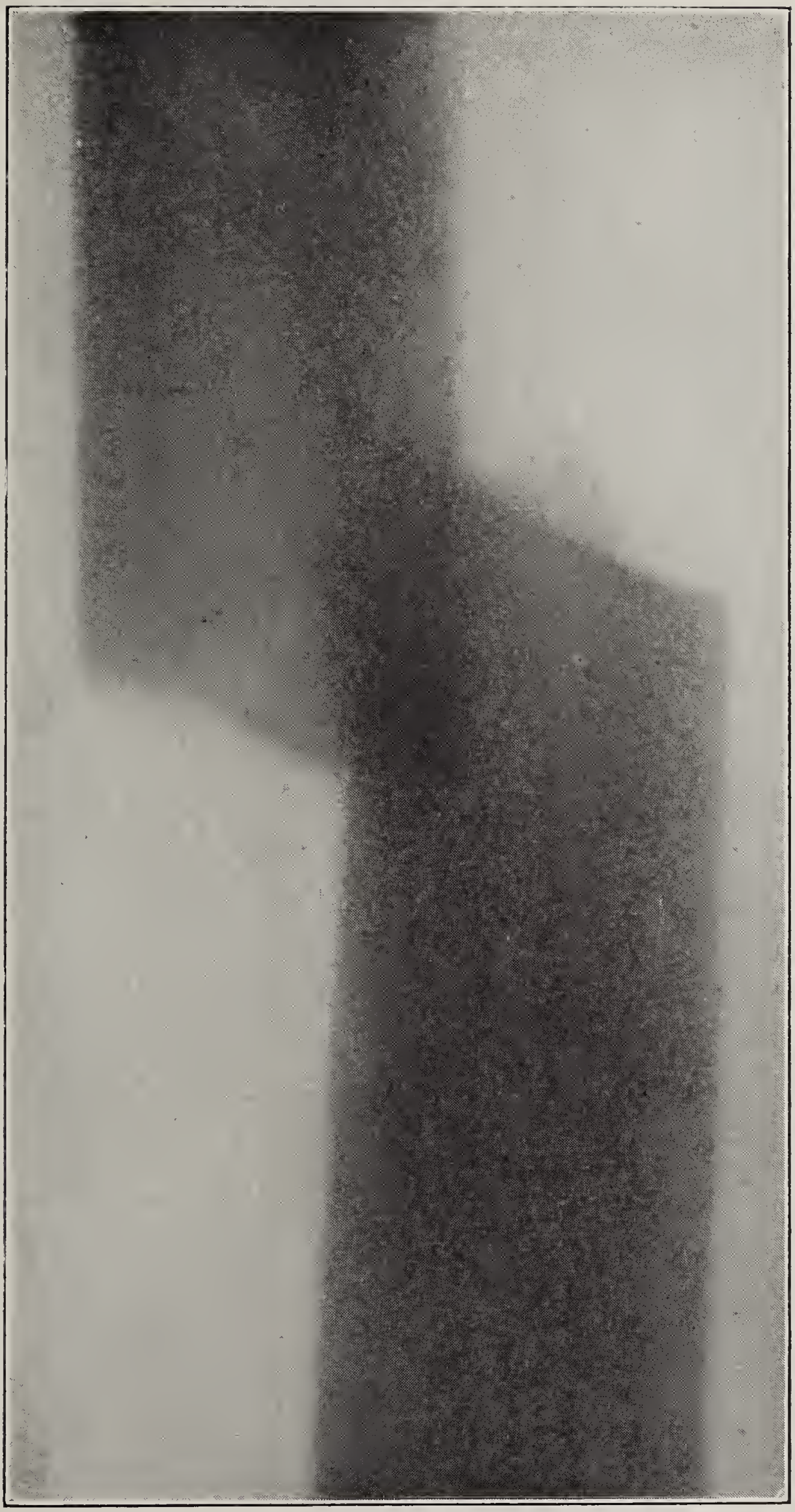

FIG. 65.—Case II, fracture of femur, irreducible without operation.

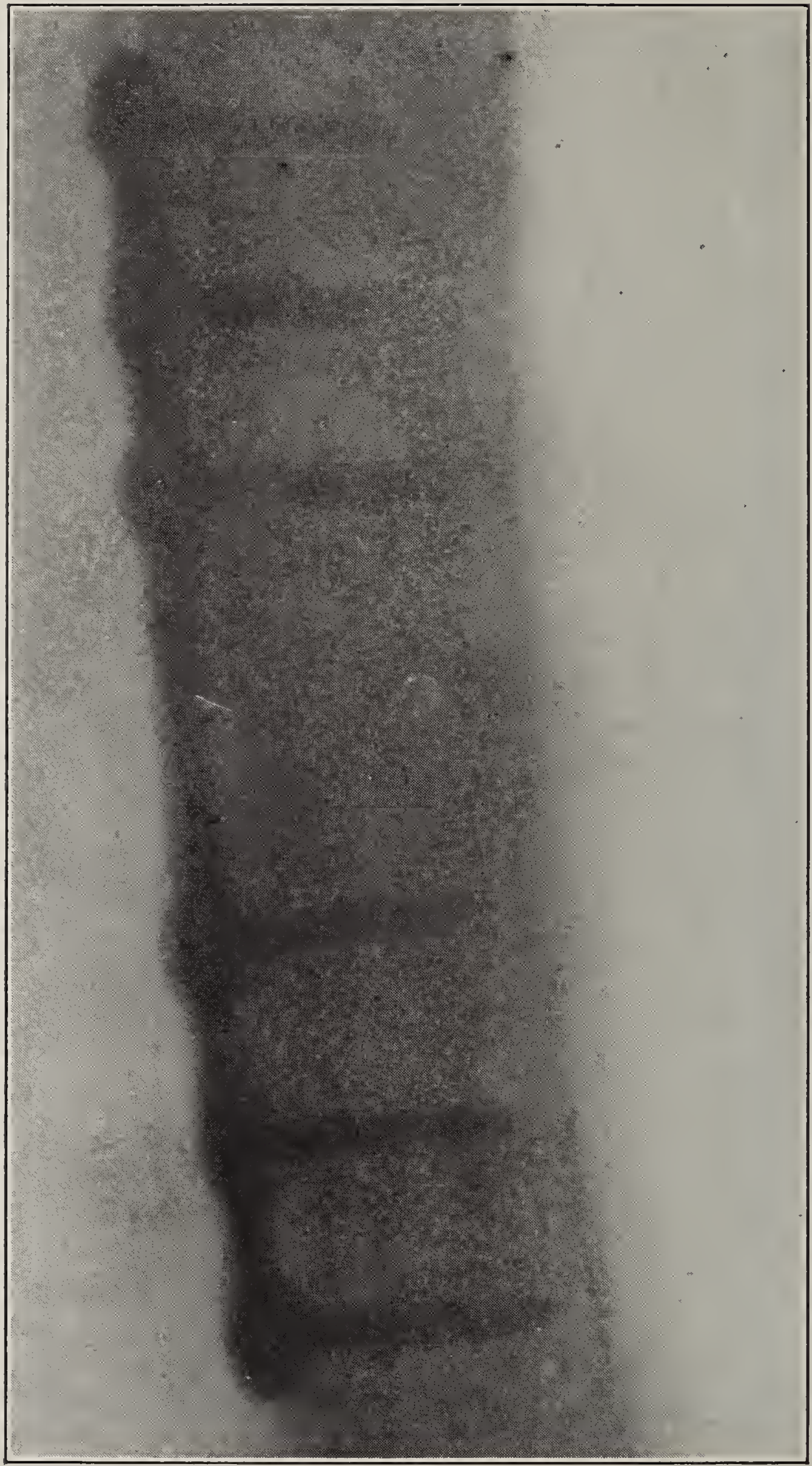

Fig. 66.—Case II, fracture reduced and plated.

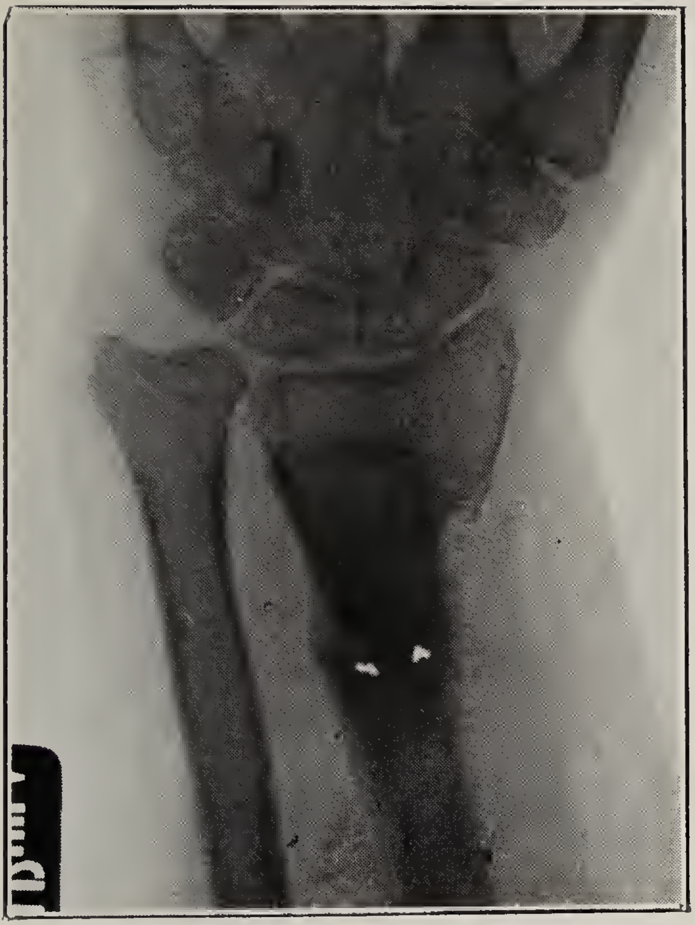

FIG. 67.—Case III, unreduced Colles's fracture, before second operation.

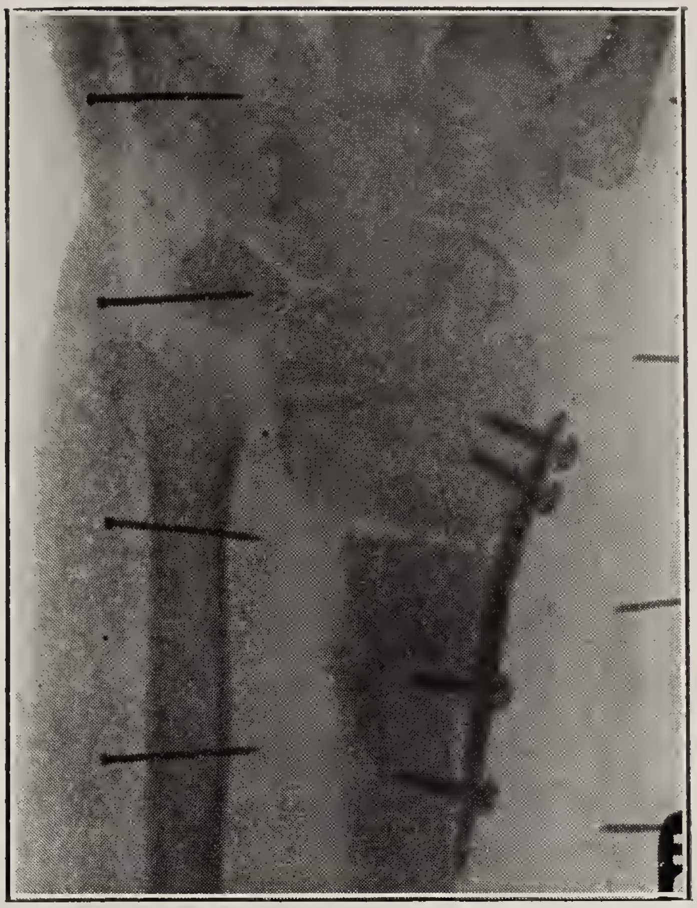

FIG. 68.—Case III, condition after operation.

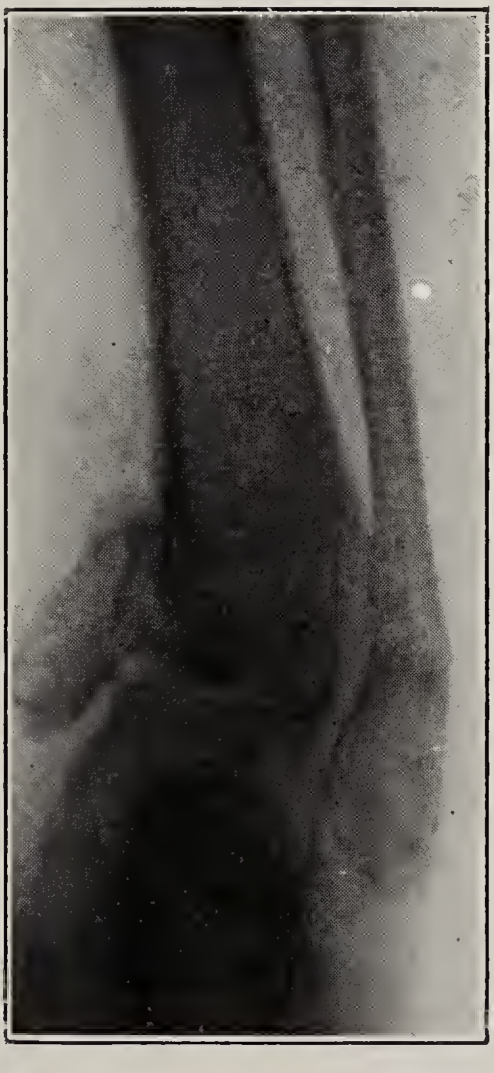

FIG. 69.—Case IV, malleolar fracture of ankle, before operation.

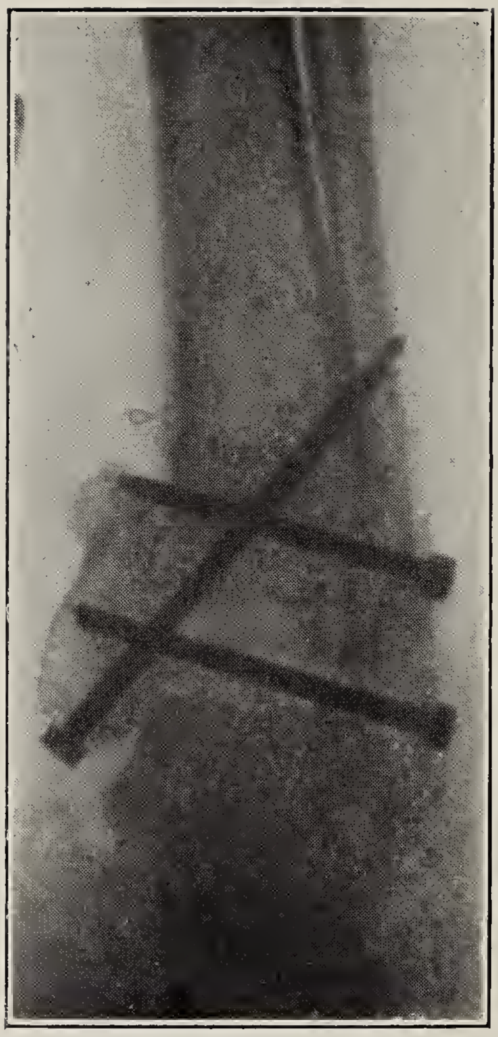

Fig. 70.—Case IV, after operation.

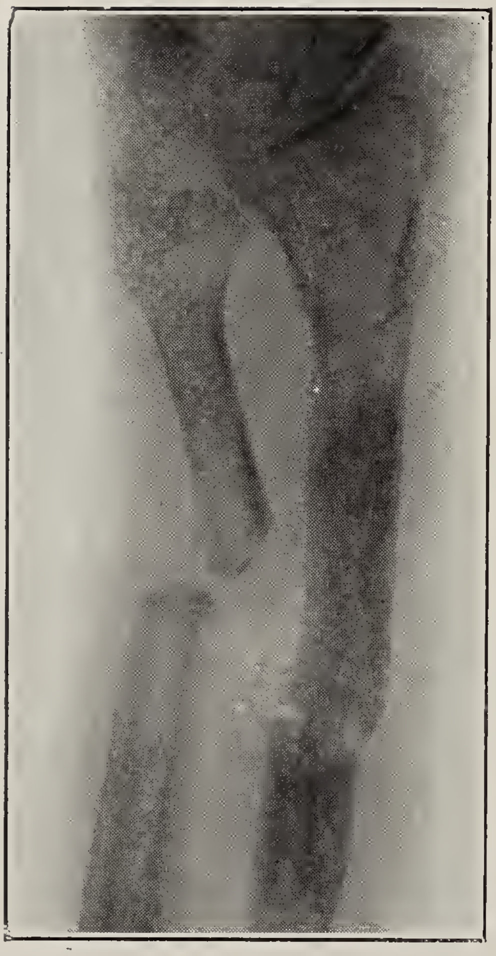

Fig. 71.—Case V, one year after injury.

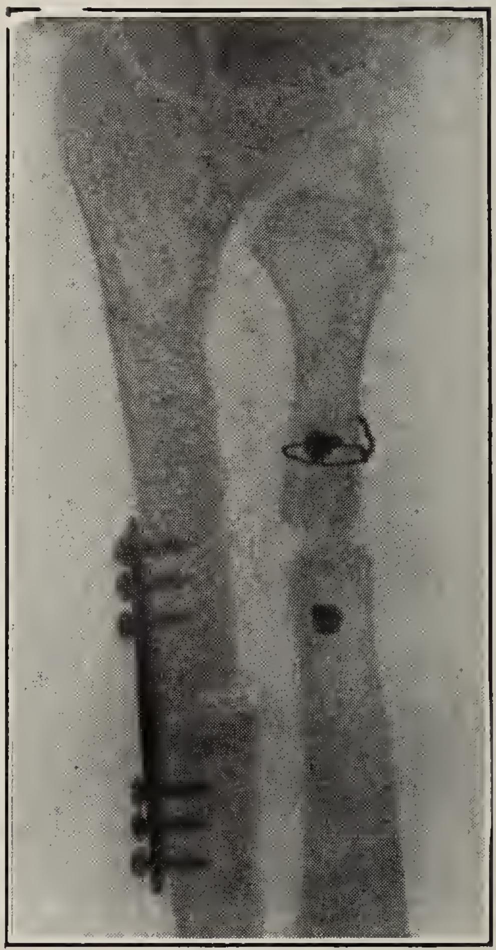

Fig. 72.—Case V, after plating and transplanting.

dressed in abduction. The fractures of the forearm and the femur were also reduced while the patient was under the anesthetic, and very satisfactory positions secured. Convalescence was uneventful. Within two weeks time the fractures of the humerus and forearm had united, and union was progressing in the femur.

If sufficient reduction is not possible by manipulation even under an anesthetic, and with all the aids known to surgery, it is proper to resort to open operation.

*Fracture of the Shaft of Left Femur, Irreducible without Operation; Fixation by Plate.*

Case II.—Robert F., aged twenty-three years, first came under my care in Dr. Frazier's service at the Episcopal Hospital on August 1, 1911. Two weeks previously this patient was knocked down and run over by a wagon, sustaining a fracture of the left femur above its middle. On August 2, I had the patient etherized, and made attempts to reduce the fracture, in which there was overlapping of the fragments to the extent of one and a half inches (4 cm.). Although end-to-end apposition was not secured (Fig. 65), the shortening was reduced to one-half inch (1.25 cm.). This might have been considered sufficient reduction if the patient was old and feeble; but in a healthy young man, with his life before him, a better result evidently was demanded. Accordingly on August 7, the patient was again anesthetized, the fractured ends exposed, reduced, and fixed by plating (Fig. 66). The patient went home on crutches about two months later, and has had no further disability. There is no shortening, and free motion in the knee (75 to 180 degrees).

If fractures were properly treated from the first, subsequent operations for malunion or for non-union would be very rare. Unfortunately it is still very often necessary to undertake operative correction of such disabilities. In some cases of *malunion* simple subcutaneous osteotomy will suffice to restore the axis of the bone; or excision of exuberant callus or of the projecting ends of the fragments may bring relief. In other cases formal resection of the fractured bone or bones must be undertaken, with fixation by plate or screw.

*Unreduced Colles's Fracture of the Right Radius; Absolute Disability, Not Relieved by Subcutaneous Osteotomy. Resection and Plating.*

Case III.—Lena F., aged forty-nine years, sustained a Colles's fracture of the right radius in June, 1913. After two months of treatment she was etherized, and an attempt at reduction was made. This was not successful, marked "silver-fork" deformity and radial deviation of the hand persisting. In the autumn of 1913 this patient was referred to the orthopedic service of the Episcopal Hospital, and first came under my care. The hand was perfectly useless, and, what was even worse, was constantly painful, with referred pains up to the elbow and shoulder. For five months massage and passive movements were applied with little if any improvement. Therefore, in April, 1914, subcutaneous osteotomy of the radius was done, in the hope that restoration of the axis of the bone might restore some usefulness to her hand or at least relieve the neuritic pains. The patient felt so much better after the operation that a very few days after the splint was discontinued (four weeks after operation) she endeavored with this hand to support the head of her daughter during a convulsive attack. This gave her wrist a very severe wrench which incapacitated her for some weeks. The pains in the elbow and shoulder, however, ceased almost entirely after the osteotomy.

As considerable deformity remained, and the hand was still almost entirely useless four months after the first operation, it was determined to operate again. Fig. 67 shows the appearance of the bones at this time. There was marked radial deviation of the hand, the radius being considerably shorter than the ulna, while the carpal articulation was further disturbed by the dorsal displacement of the lower radial fragment, whose carpal surface faced toward the extensor rather than toward the flexor surface of the forearm. The healed osteotomy is visible above the fracture, with the deformity due to the unfortunate accident after the first operation.

On August 29, 1914, an open operation was done. The flexor tendons were loosened up from the fractured surfaces, the radius was divided, the fractured ends resected until proper alignment was secured, the head of the ulna was removed subperiosteally; and the radius fixed by a plate. The result is seen in Fig. 68. The normal contours of the articulating surfaces have been restored, the ulna now being no longer than the radius and the carpal surface of the radius facing forward (toward the flexor surface) instead of backward (toward the extensor surface) as formerly. Healing was delayed by some sloughing of the edge of the skin flap, but within five weeks after operation the woman could appose her thumb easily to all the fingers, and had some power of grasping in the hand. Unfortunately neuritic pains persist, and long after-treatment will be requisite.[1]

*Malunion of Bi-malleolar Fracture at the Right Ankle; Absolute Disability. Resection and Screw Fixation.*

Case IV.—Catharine R., aged forty years, sustained an inversion fracture of both malleoli at the right ankle in January, 1914. In June, 1914, she came to the orthopedic service of the Episcopal Hospital because she was still obliged to use crutches, pain preventing her from bearing any weight whatever on her foot. Fig. 69 shows the position of the bones at this time. It was evidently hopeless to look for improvement when none whatever had occurred in five months, and such marked deformity was present. Accordingly, on June 27, 1914, I exposed the fractures of both tibia and fibula, separated the fragments by chisel, along the lines of fracture, reduced the deformity, and fixed the bones in proper position with screws. No retentive dressing was required, owing to the security of the screw fixation. This enabled light passive motion to be instituted the day after operation, and within ten days, when the wounds had healed, no pain or disability remained. The patient was forbidden to bear any weight on her foot for more than two months. At the end of this time, on making the first attempt to walk, she threw away her crutches and walked painlessly, though with a slight limp from stiffness. Last week, she tells me, she visited Mauch Chunk, and after a ride on the "Switchback" walked all the way down the side of the mountain without difficulty. Fig. 70 shows the position of the bones after operation.

In the case of both the patients whose histories have just been reported (Cases III and IV), the proper time for operation was soon after the injury, when it was found impossible to secure reduction of the fragments. In both cases many months of discomfort would have been saved, and the ultimate result would almost certainly have been better than with delayed operation.

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[1] The plate was removed January 20, 1915, and in February the patient commenced to have fair use of the hand, with less pain.

*Non-union* of a fractured bone presents even a more difficult problem than malunion. Resection and fixation by plates or screws do not always succeed in arousing sufficient osteogenesis to secure firm union. In many cases it is better to bridge the gap by a bone transplant, taken from the same bone or elsewhere in the patient's body. I have reported before the Philadelphia Academy of Surgery (*Annals of Surgery*, 1914, ii, 779) the very satisfactory result obtained by bone transplantation in a case of ununited fracture of the neck of the femur, and give below the notes of another case of non-union in which transplantation of bone was employed.

*Ununited Fracture of Radius and Ulna; Fixation by Bone Transplant and Steel Plate.*

Case V.—Fred. H., aged thirty-five years, on May 27, 1913, was caught in shafting and swung around the machinery in his mill until the power could be shut off. He was admitted at once to the Episcopal Hospital. He sustained a compound fracture of both bones of the right forearm, all four bone ends projecting through the soft parts; as well as a compound double fracture of the humerus of the same arm, one of the fragments here also projecting through the skin. I cleansed the wounds thoroughly, repaired the damage to the soft parts and reduced the fractures as far as possible. The humerus healed by firm bony union without appreciable deformity; but though healing of the soft parts in the forearm occurred as soon as could be expected, the bones failed to unite. They maintained fair position, but the ulna, part of which had to be excised at the time of the accident, remained entirely ununited; while only fibrous union was secured in the radius (Fig. 71). Operation to remedy this non-union was done September 9, 1914 (sixteen months after the original injury): the radius was resected until healthy marrow was exposed, and then plated; while the ulnar fragments, the gap between whose ends could not be bridged without sacrificing an undue length of the radius, were united by a transplant cut from the ulna higher up. Fig. 72 is from a skiagraph made a few days after operation. Though bony union has not taken place in either radius or ulna at the end of six months, firm fibrous union has been secured, and the usefulness and stability of the arm are markedly increased.

Treatment of Ankylosis. Ankylosis was formerly treated by excision of the joint affected. The classical excision of a joint such as the elbow or shoulder, was designed to secure a movable joint; in the knee, on the other hand, excision was adopted only when the joint was ankylosed in bad position, and aimed to secure a stiff joint in good position. The modern operation of *arthroplasty* (by interposition of fat and fascia flaps) enables the patient to secure not only much greater stability at the elbow and shoulder than was secured by excision, but even at the knee and hip a fair degree of motion may be secured without impairing the stability of these joints. This is of especial value at the knee, as a stiff hip is more readily compensated for; but the operation is difficult, and not always successful. Probably if a surgeon obtains 50 per cent. of successful results in his arthroplasties at the knee he will be doing well. Even if a full range of motion is not secured, the patient usually is satisfied to obtain enough motion to promote walking and to enable him to be seated comfortably. I select the following case for report here, because the operation of arthroplasty was done at both knee and elbow in the same patient.

*Ankylosis of Elbow and Knee; Arthroplasty.*

CASE VI.—Gertrude T., aged twenty-three years, applied to Dr. Harte's service at the Orthopædic Hospital on March 2, 1913, for ankylosis of the left elbow and the right knee. In May, 1912, when about seven months pregnant, but without any evident cause (such as preceding tonsillitis, influenza, etc.), this patient had felt pain and stiffness around her right hip during one entire day. That night she was awakened by pain in the left elbow, and next morning she found it swollen. The second night the right knee became painful and swollen. She was not confined to bed until the fourth day, and was sent to the Philadelphia Hospital on the sixth day of illness. She remained in bed in the hospital for seven weeks, with fever, and acute arthritis. The pregnancy was terminated normally after convalescence.

When first seen at the Orthopædic Hospital, ten months after the onset of the arthritis, this young woman was unable to walk without crutches. There was about ten degrees of motion in the knee (130 to 140 degrees), which was not painful. The patella was ankylosed to the condyles of the femur, but the femur and tibia were not united by bony ankylosis. At operation, however, it was found impossible to secure more than 10 degrees of motion in the knee even after the patella had been loosened from the condyles until the condyles themselves were resected; evidently the arthritis had distorted the articular surfaces so greatly as to prevent motion even though no bony ankylosis between tibia and femur was present.

The elbow was fixed in bony ankylosis at an angle of 110 degrees; fortunately the radio-humeral and the upper radio-ulnar joints were not involved, as rotation in the forearm was normal.

Arthroplasty of the elbow was done May 1, 1913, and of the knee three weeks later. Convalescence was uneventful. Free motion was secured in the elbow (50 to 160 degrees), and fair motion in the knee (120 to 175 degrees) by the time she was discharged from the hospital, three months after the last operation. At this time she was able to walk without crutches. Fig. 73 shows the condition on admission, Figs. 74 and 75 show respectively the amount of flexion and of extension present on discharge.

The patient was seen again one year after operation, when she returned to the hospital for a sprain of the knee which had been operated on, due to a recent fall. Until this injury she had been able to walk about well without any support, going up and down stairs constantly and doing her own housework and caring for her children. There was at this time free motion in the elbow (45 to 150 degrees), and in the recently sprained knee there was motion from 95 to 150 degrees. As there was still some effusion in the knee from the recent injury, and motion was painful, it is probable that the full range of motion was not secured at this examination.

Examination October 17, 1914, showed free motion in the elbow, from 45 to 150 degrees; there was active power of extension in the triceps, and the joint was quite stable. In the knee there was motion from 100 to 167 degrees, but although the patient could stand momentarily on this leg without other support, the power in the quadriceps was not sufficient to maintain the knee in extension if the limb was held up horizontally. In the house she walks without support, but uses a cane on the street. She cannot go up stairs "leg over leg." She claims that the knee is still gaining strength, and that it is now much more useful than six months ago.

Bone Transplantation. One of the chief indications for this operation, as already mentioned, is non-union after

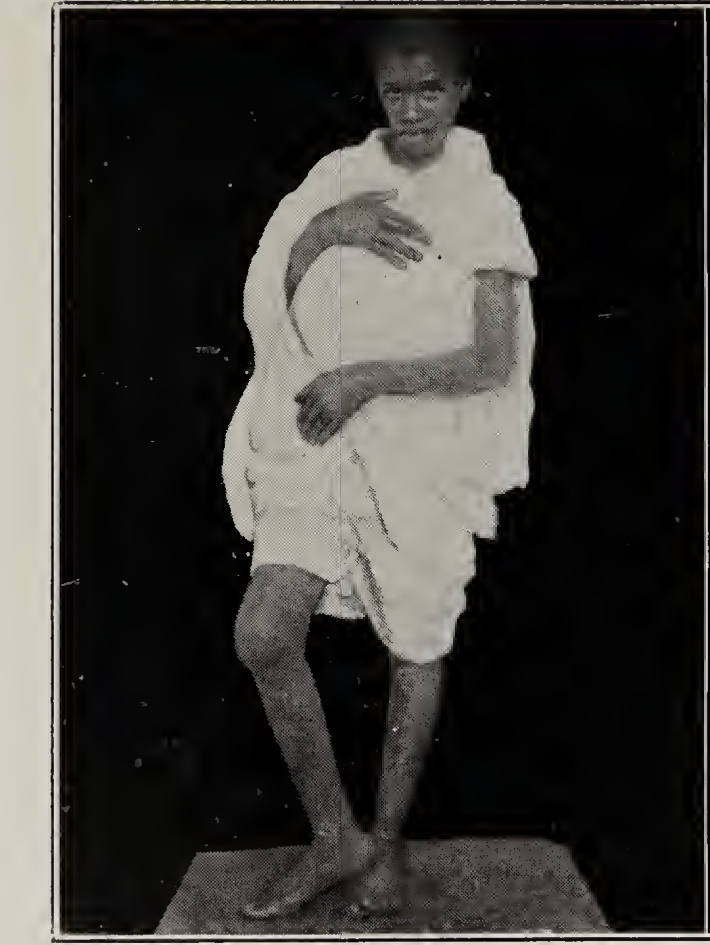

Fig. 73.—Case VI, condition on admission.

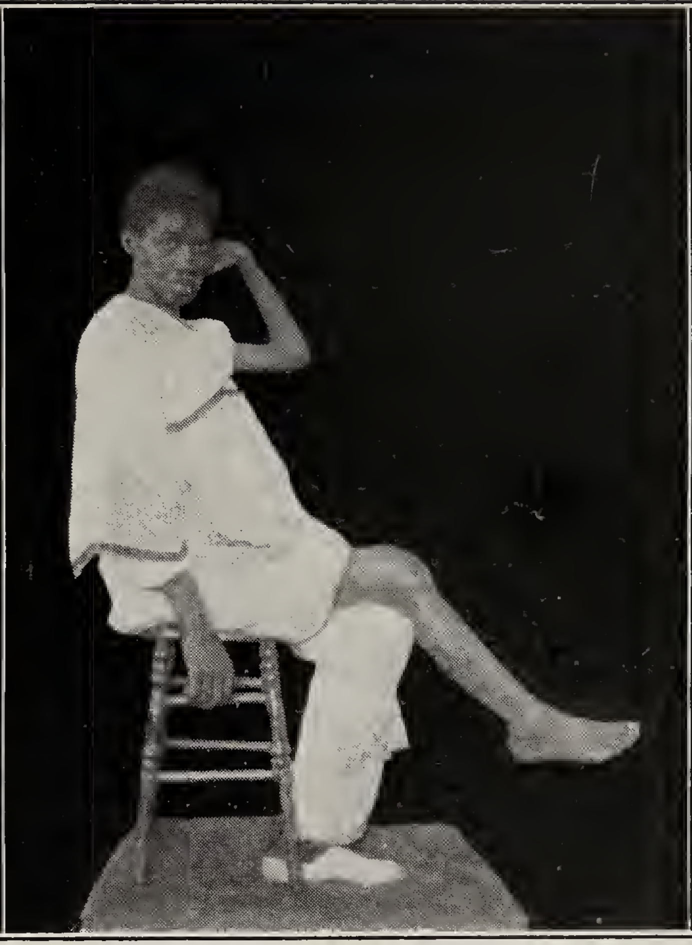

Fig. 74.—Case VI, limits of flexion three months after operation.

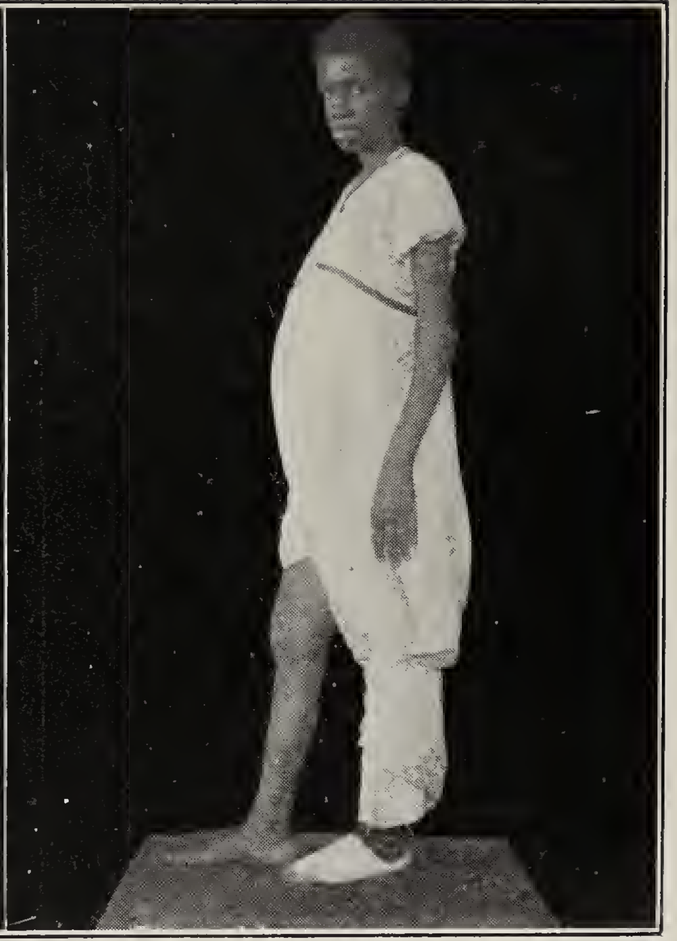

Fig. 75.—Case VI, limits of extension three months after operation.

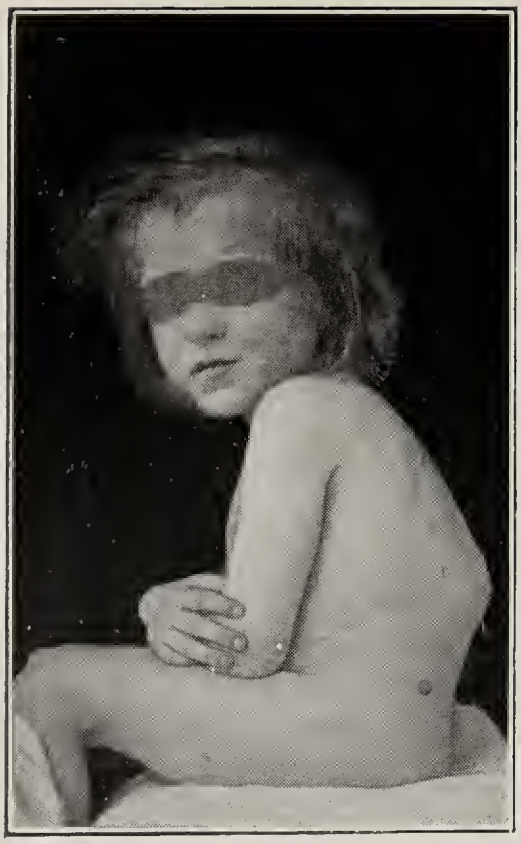

Fig. 76.—Case VII, tuberculosis of spine on admission.

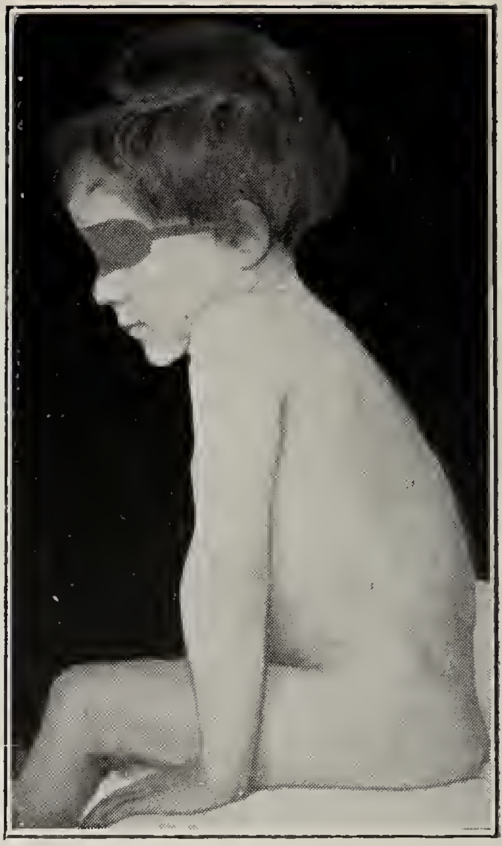

Fig. 77.—Case VII, result of bone transplantation.

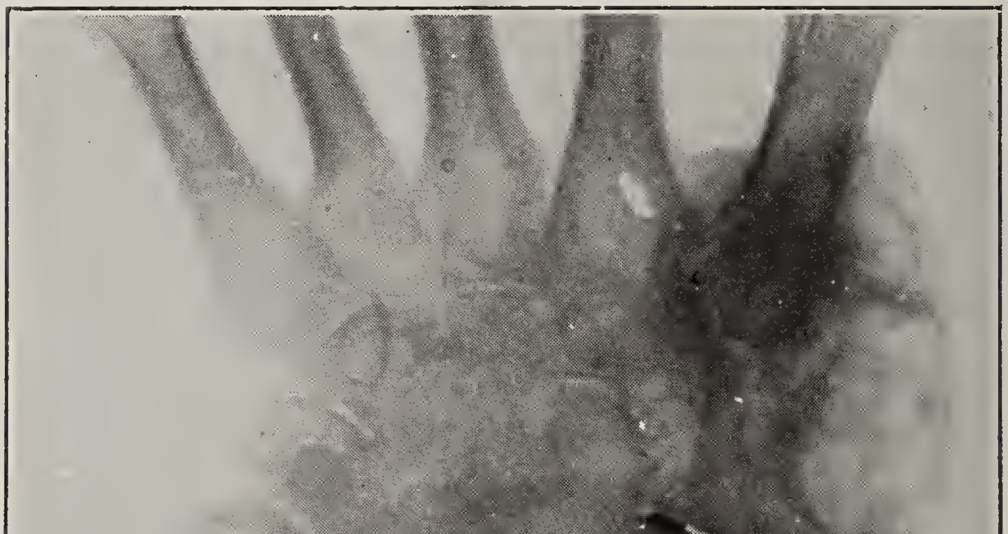

Fig. 78.—Case VIII, destruction of lower end of radius by "giant-celled sarcoma."

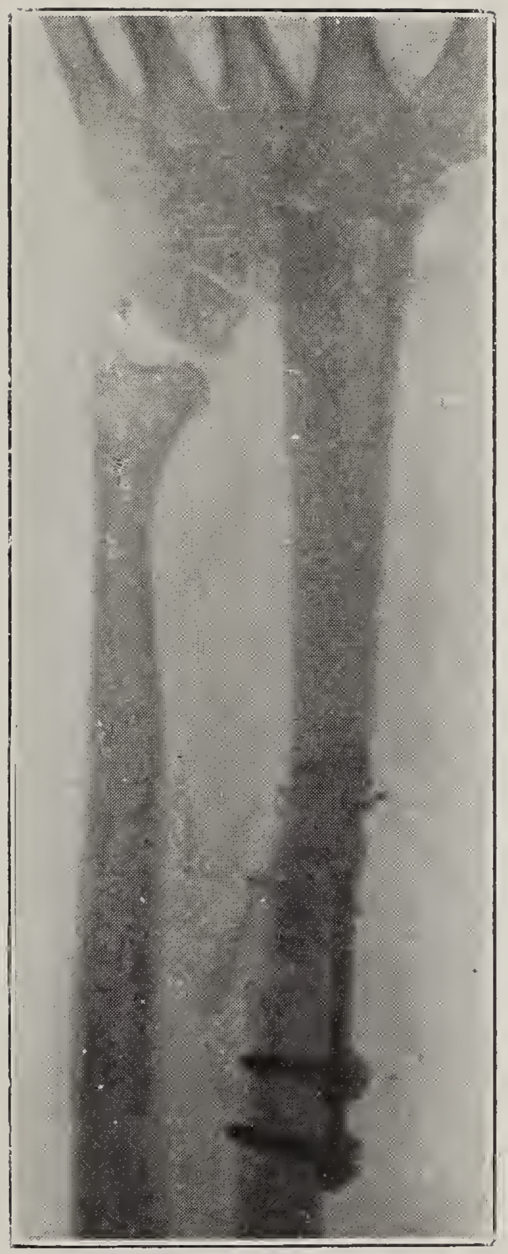

FIG. 79.—Case VIII, result of bone transplantation.

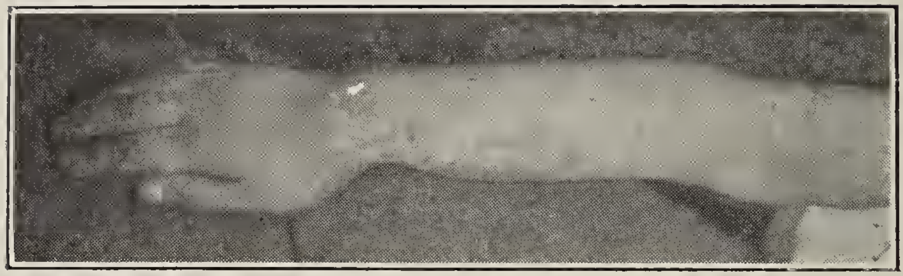

Fig. 80.—Case VIII, three months after operation.

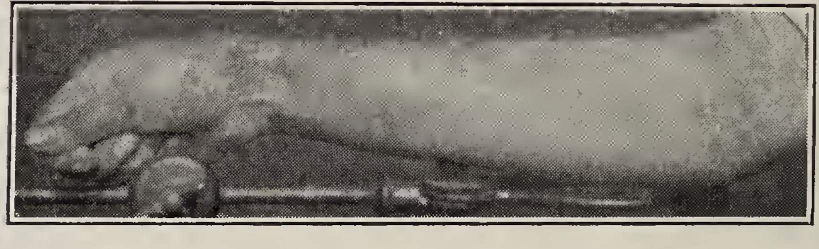

Fig. 81.—Case VIII, three months after operation.

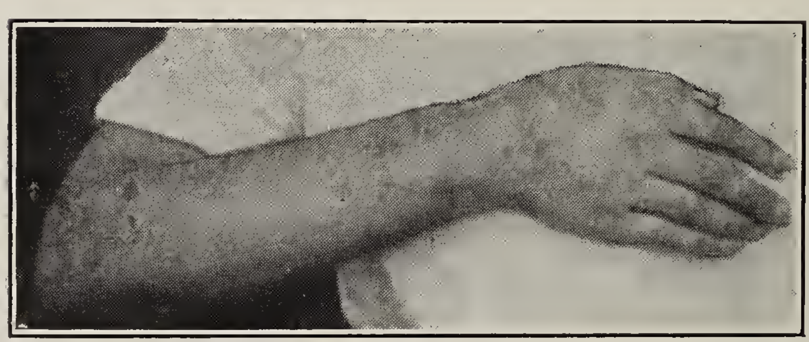

Fig. 82.—Case VIII, three months after operation.

fracture. It is even more frequently employed to secure fixation of the spine in cases of tuberculosis of the vertebræ; and after excision of bone tumors the insert of a transplant may be the only satisfactory way to maintain the form of the limb.

*Tuberculosis of Vertebræ; Fixation by a Bone Transplant.*

CASE VII.—Emma C., aged four years, was admitted to my service at the Episcopal Hospital, May 18, 1914, with tuberculosis of the spine of several months' duration, hitherto untreated (Fig. 76). After one month's recumbency on a Bradford frame the deformity had markedly decreased, and on June 27, 1914, a transplant was cut from the patient's tibia and inserted in the split spinous processes of the 11th and 12th thoracic and the 1st, 2d, 3d, 4th, and 5th lumbar vertebræ. This almost completely reduced the kyphos. Seven weeks later, however, it was found that the lower end of the transplant projected unduly beneath the skin, and a skiagraph showed that it had sprung loose from the 4th and 5th lumbar vertebræ. Accordingly, on August 15, 1914, after nicking the transplant with a saw, a green-stick fracture was produced in it just below the spine of the 3d lumbar vertebræ, and the end of the transplant was bent inward and embedded again in the spinous processes of the 4th and 5th lumbar vertebræ. Six weeks after the last operation the deformity was inconspicuous and the child's health perfect (Fig. 77).

The object in fixing the spine by a bone transplant of course is to cure the disease by immobilizing the spine more thoroughly than can be accomplished in any other way. Incidentally this method prevents recurrence of deformity, or at least prevents increase of deformity if that which has already developed cannot be overcome by recumbency before operation. It is my practice to put these patients to bed until the deformity has nearly or quite disappeared, and until they learn to lie reasonably quiet; then to operate, and to keep them in bed about six weeks after operation. They are then allowed to be about with a light brace, which is worn for about six months. Many patients, however, discard the brace contrary to advice very soon after operation. But though I have never seen any evil consequences, I believe it is safer to wear a support for the time indicated. The operation in itself appears to be devoid of serious risk. I say this notwithstanding the fact that the first patient on whom I employed it died of pneumonia, and that another patient died of tuberculous meningitis about seven weeks after operation. A patient *may* die of pneumonia after any operation; and many a patient with tuberculosis of bone who has had no operation develops tuberculous meningitis while lying quietly in bed. So that these fatal issues are not, I believe, to be charged to the operation itself. It is noticeable that even very soon after the operation the patients improve in health and seem to feel much stronger.

*Myeloma of Radius; Excision and Transplantation of Bone.*

CASE VIII.—Sophy M., aged twenty-two years, applied to Dr. Harte's service at the Orthopædic Hospital, February 20, 1913, with a cystic swelling of the wrist, which at first sight was mistaken for tuberculosis of the tendon sheaths. The history was that the swelling had gradually developed during the preceding year (since February, 1912) from no well ascertained cause. A skiagraph (Fig. 78) showed the lower end of the radius destroyed by a cyst, which, however, left the wrist-joint and the overlying soft parts intact. On March 5, 1913, Dr. Harte opened and curetted the cyst, whose walls were very thin, there being scarcely any trace of bony tissue discernible.[1] A transplant was cut from the tibia and inserted in the interior of the cyst to maintain the form of the bone and in the hope of stimulating osteogenesis. For months the local condition continued to improve. The tumor became smaller and its walls firmer; it lost its cystic feel, but the forearm remained abnormally large, and caused the patient considerable annoyance. A skiagraph made September 24, 1913 (six months after operation), showed that the transplant was being absorbed. Early in 1914 the tumor again began to soften, the local heat and discomfort increased, until finally in June, 1914, the girl consented to another operation. She was referred to my service at the Episcopal Hospital. On June 27, 1914, I excised the entire lower end of the radius, including its periosteum and articular surface.[2] It was found that the extensor tendons of the thumb passed through a canal in the tumor, and while one of the tendons could be pulled out of this canal after being divided, the other could not be extracted and a couple of inches of it had to be removed with the tumor. Moreover, the tumor so overhung the carpal bones, although it had not broken through the articular surface of the radius, that it was impossible during the operation to outline its limits precisely, and the scaphoid and semilunar bones were inadvertently removed along with the tumor. A transplant was then cut from the tibia (5 inches long, and of the thickness and shape of the lower end of the radius) and inserted in the large wound caused by removal of the tumor. This transplant was fixed to the shaft of the radius by a steel plate; the plate was screwed to the radius, but as the screws fixing it to the transplant twisted loose when half inserted, it was necessary to fix the plate to the transplant by a wire loop (Fig. 79). Healing was uneventful. A splint was worn for three months after the operation. At present the transplant is firmly united to the shaft of the radius; there is rotation in the forearm through an arc of more than 90 degrees; there is flexion in the wrist to 140 degrees, and extension to a straight line (180 degrees). The girl can make a good fist, and has fair power of grasping which is rapidly improving. Figs. 80, 81, and 82 are from photographs made three months after operation.[1]

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[1] Dr. Paul A. Lewis, of the Pennsylvania Hospital, reported that the growth was a "giant-celled sarcoma."

[2] Dr. C. Y. White, Director of the Pathological Laboratories of the Episcopal Hospital, reported that the growth was a "giant-celled sarcoma."

Many other examples could be cited, exemplifying the value of modern surgical procedures in different lesions of the bones and joints. Nothing has been said of the treatment of dislocations, otherwise irreducible, by arthrotomy, reefing of the capsule, etc.; of the treatment of osteomyelitis by excision of the diaphysis, which gives admirable results in selected cases; nor of the treatment of paralytic flail-joints by production of ankylosis (arthrodesis), which is an operation of the utmost value. The cases herein reported serve merely as samples of the kind of work that is now being done, and I trust they may stimulate the interest of the profession in a class of patients too often neglected, or in whom the most favorable time for operative correction of the disability is allowed to pass before the surgeon is consulted.

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[1] Two years after operation the condition remains satisfactory.

# ARTHROPLASTY OF THE ELBOW[1]

By ASTLEY PASTON COOPER ASHHURST, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

I HAVE adopted arthroplasty of the elbow-joint in five patients: twice for bony ankylosis, and three times for marked limitation of motion following fracture.

TECHNIQUE OF THE OPERATION. 1. *Eposure of the Joint.* The skin incision (Fig. 83)[2] begins on the external supracondylar ridge of the humerus, about 5 cm. above the joint, and is continued straight downward to the joint level where it is curved slightly backward toward the extensor surface of the forearm; its entire length is about 10 cm. This incision is carried down to the supracondylar ridge above the joint; below the joint level the deep fascia is exposed but is not incised. The soft parts are then cleared from the humerus, the brachioradialis and the extensor carpi radialis longior are displaced forward and the triceps backward, *thoroughly* exposing the external condyle, the anterior capsule of the joint, and the external lateral ligament with the origin of the extensor muscles (Fig. 84). The external condyle is then detached from the humerus by osteotome, the bone section entering the elbow-joint on the capitellar surface of the humerus. In most cases even when the ulnohumeral joint is ankylosed the radiohumeral joint is free, and the external condyle may be easily turned downward on the external lateral ligament as a hinge, exposing the joint (Fig. 85). If ankylosis is present between the radius and humerus it is easy to separate them by gouge without injury to the external lateral ligament. In order to turn the condyle downward sufficiently to expose the joint thoroughly, the capsule must be snipped with scissors in front of and behind the external lateral ligament.

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[1] Read before the Philadelphia Academy of Surgery, April 5, 1915. Reprinted from Annals of Surgery, 1915, ii, 303.

[2] The accompanying illustrations (Figs. 83 to 92) are from photographs of preparations in the Laboratory of Operative Surgery in the University of Pennsylvania.

2. *Dislocation of the Joint.* If ankylosis exists between the ulna and humerus, these bones are separated by a suitably shaped gouge, driven transversely across the joint by smart blows from a hammer. When the union has been almost completely divided, the remaining fibres on the inner side of the joint may be ruptured by abrupt, short, forceful movements of flexion and extension applied to the elbow joint. If one fears injury to the ulnar nerve a small incision may be made over its course between epitrochlea and olecranon, and the nerve may be drawn away from the bones. Only in one case did I find this necessary.

Ankylosis having been overcome, or in cases where no ankylosis is present, the elbow joint is dislocated by adducting the forearm around the internal lateral ligament, as a hinge, until the forearm lies almost parallel with the upper arm, causing the ends of the humerus, radius and ulna to come into full view (Fig. 86).

3. *Shaping the Bone Ends.* As little as possible is done to the ulna, especially when the head of the radius is healthy. Reliance is placed on resection of the humerus for shaping the new joint (Fig. 87). If ankylosis exists in the upper radio-ulnar joint, it is simpler to resect the head of the radius than to turn in a flap between radius and ulna. For shaping the humerus a Gigli wire saw mounted in a bow-shaped frame (the saw of Pierre Delbet) is the most convenient instrument; with this a series of cylindrical sections can be removed from the humerus until enough room is secured between the bone ends. Seldom is it necessary to remove any bone above the level of attachment of the internal lateral ligament at the base of the epitrochlea.

4. *Interposition of the Flap.* The bones being temporarily restored to their normal relations, the original skin incision is extended backward from its upper end across the posterior surface of the arm (Fig. 88). The triangular skin flap thus outlined is raised, including a fair amount of subcutaneous fat, until the superficial surface of the triceps, or of the fat and fascia covering it, is fully exposed. An interposing flap of fat and fascia is then raised from the superficial surface of the triceps, with its base at the olecranon (Fig. 89). It is best to include some of the triceps aponeurosis and muscular fibers in this flap. The elbow joint is then partially dislocated again, and the flap is attached to the internal lateral ligament of the elbow, and to the anterior and posterior capsules of the joint, by a few interrupted sutures of chromicized catgut (No. 0), thoroughly covering the articular surface of the humerus (Fig. 90).

5. *Closure of the Wound.* The forearm is restored to its normal relation with the arm, and the external condyle is brought up in front of the pedicle of the interposing flap, and is fixed to the humerus (Fig. 91). For this purpose I prefer Lambotte's self-boring screws; in the accompanying illustration a nail was employed because at the time (in the Laboratory of Operative Surgery) no such screws were at hand. I have also used chromic gut and phosphor-bronze wire sutures, but have found them inferior to the Lambotte screws in obtaining secure fixation. Two screws are better than one. If much bone has been removed from the humerus, it will be necessary to trim the external condyle to fit.

The triceps is then sutured accurately to the brachioradialis and extensor muscles, the deep and superficial fasciæ are accurately approximated, and finally the skin wound is closed (Fig. 92). No drainage is necessary. Interrupted chromic gut sutures (No. 1 or No. 2) are employed throughout. Rarely is a single ligature required.

The average time I have consumed in the operation is about one hour and thirty minutes.

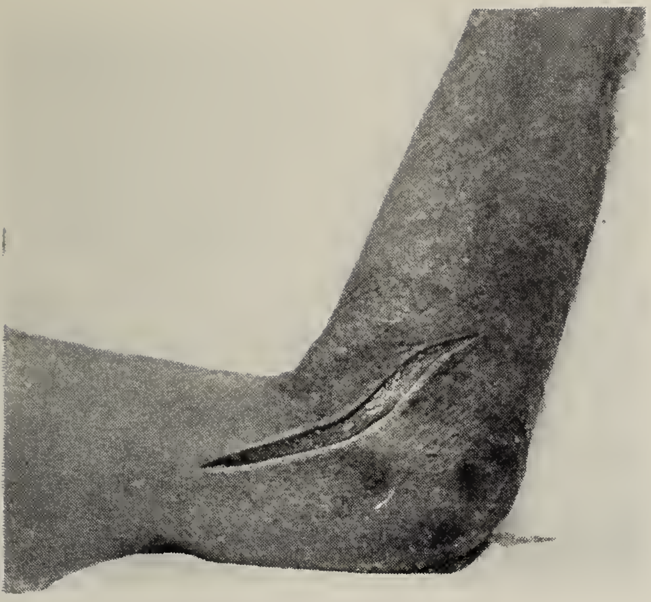


FIG. 83.—Arthroplasty of elbow; skin incision.

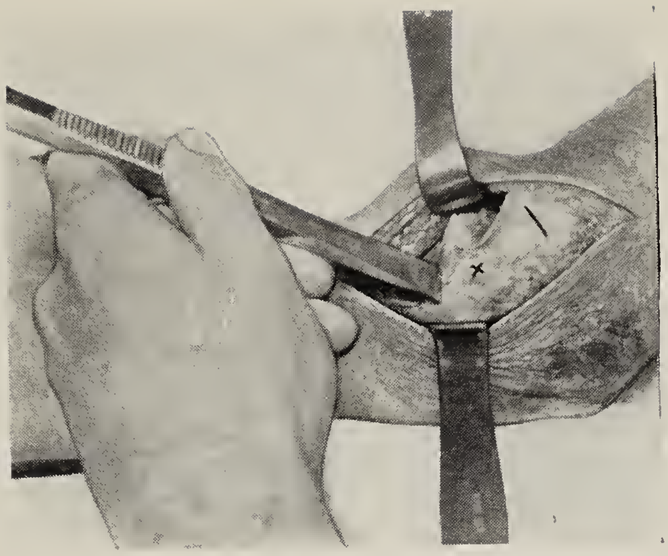


FIG. 84.—Arthroplasty of elbow; external condyle (×) and head of radius (—) exposed, and osteotome applied to external condyle.

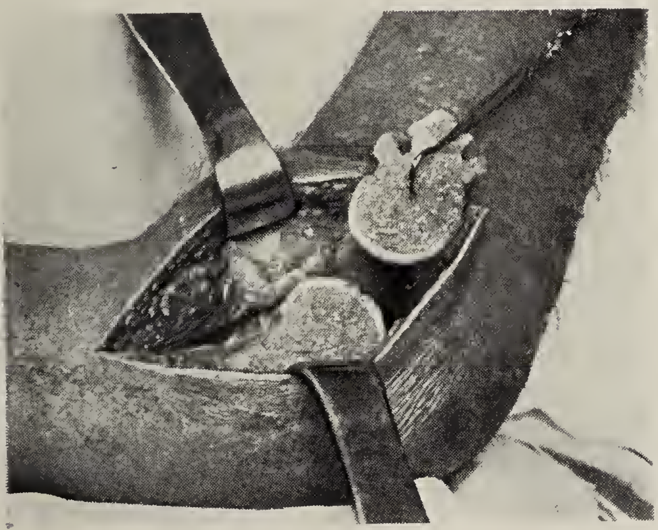


FIG. 85.—Arthroplasty of elbow; external condyle turned down, exposing joint.

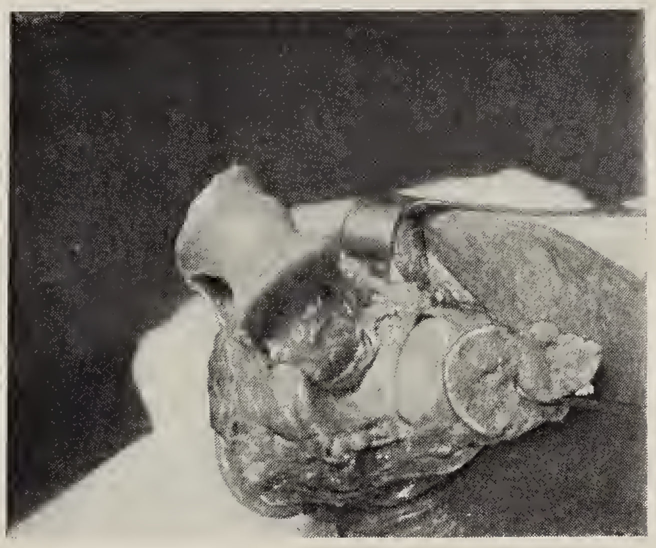


FIG. 86.—Arthroplasty of elbow; joint luxated around internal lateral ligament as a hinge.

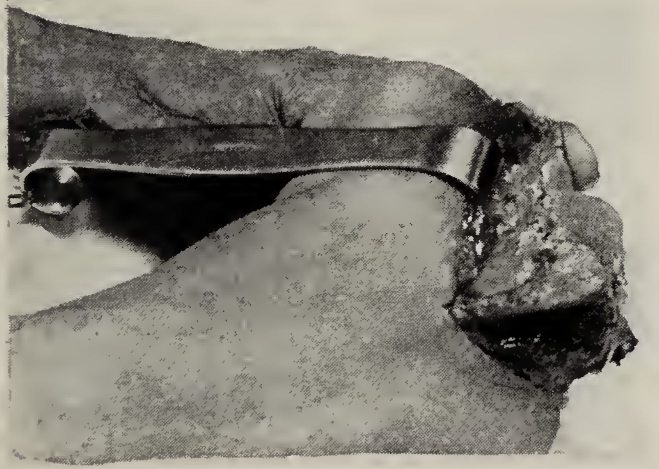


FIG. 87.—Arthroplasty of elbow; articulating surface of humerus removed with saw. Joint is viewed from outer side; external supracondylar ridge and surface from which the external condyle has been detached face the reader, and the joint surface of the humerus (freshly sawn) is directed toward the right of the picture.

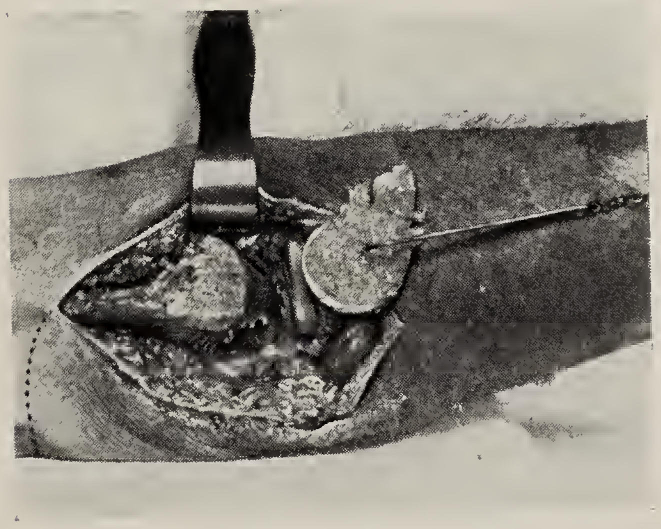


FIG. 88.—Arthroplasty of elbow; bones replaced; dotted line indicates extension of primary skin incision, to expose triceps.

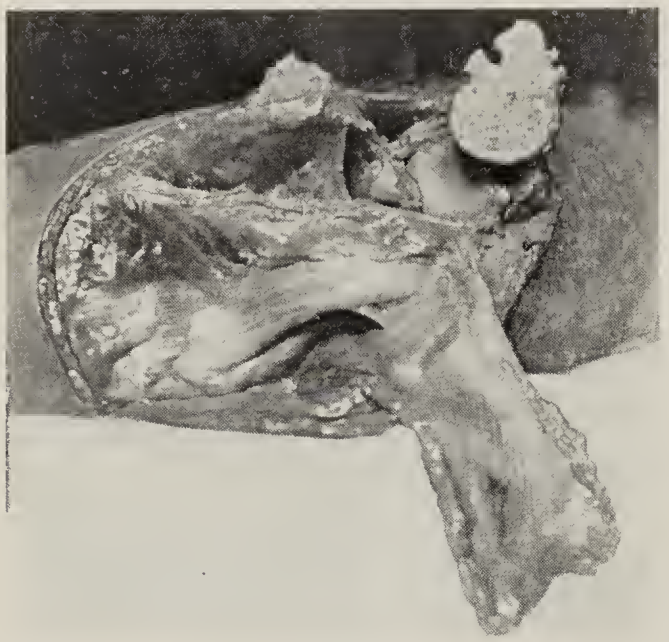


FIG. 89.—Arthroplasty of elbow; fat and fascia pedicled flap cut from surface of triceps.

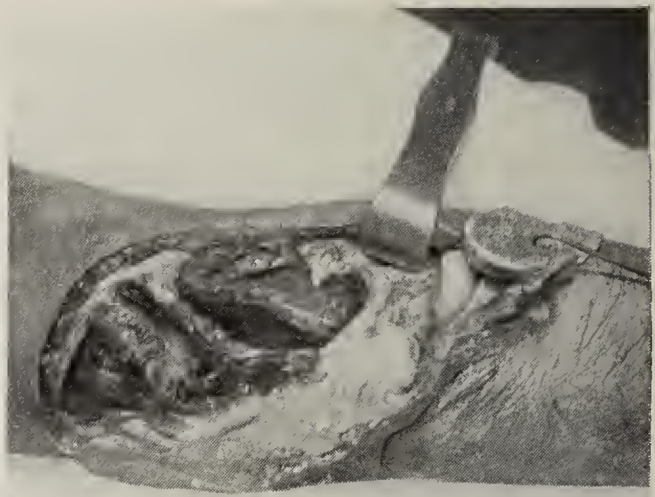


FIG. 90.—Arthroplasty of elbow; flap turned into joint covering articular surface of humerus. Same view of joint as Fig. 88.

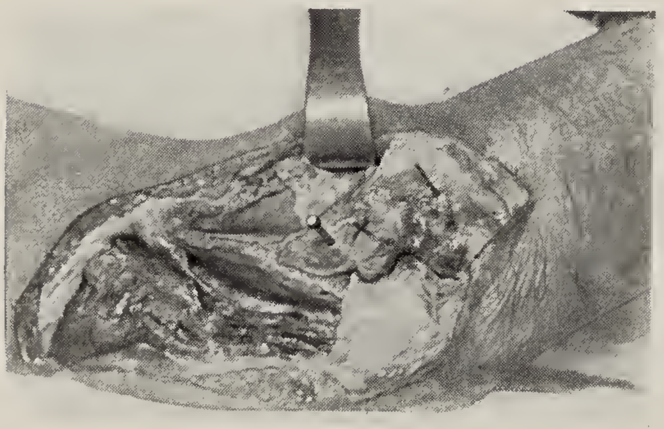


FIG. 91.—Arthroplasty of elbow; external condyle has been replaced and fastened by a screw or nail.

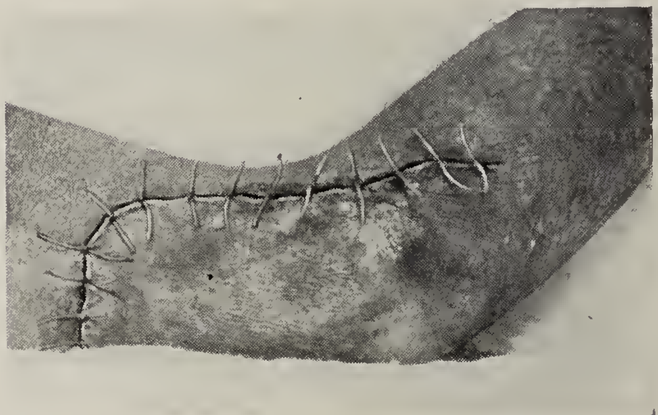


FIG. 92.—Arthroplasty of elbow; skin sutured.

## CASE HISTORIES

Case I.—*Malunion of fracture of external condyle; limited motion and cubitus varus.* James W., aged five years. Treated in Dr. Frazier's service at the Episcopal Hospital. Fractured the external condyle of his right humerus in July, 1908; and first came under my care in October, 1908, for limited motion (50 to 145 degrees) and cubitus varus (200 degrees). A skiagraph showed a fracture with outward rotation of the external condyle, but bony union. For six weeks subsequently light massage and passive movements were employed, but the range of motion improved only 10 degrees in flexion (40 to 145 degrees). Fig. 93A gives a photograph taken before operation.

*Operation* (November 18, 1908). Usual external incision. Enough of the external condyle was removed (without detaching it from the humerus) to permit full extension of the elbow, as well as to overcome the cubitus varus. The olecranon fossa on the posterior surface of the humerus was also deepened. A fatty fascial flap from the superficial surface of the triceps was turned in over the denuded external condyle and the wound closed. The elbow was dressed in hyperflexion.

At the first dressing, ten days later, the wound was healed and the skin sutures absorbed. Motion was free and painless from 45 to 90 degrees. The arm was now carried in a sling. On December 5, there was motion from 40 to 140 degrees.

In May, 1912, three years and a half after operation, the boy was presented at a meeting of the Philadelphia Academy of Surgery, exhibiting perfect function, no varus deformity, full flexion, but extension only to 150 degrees (*Annals of Surgery*, 1912, ii, 647). Fig. 93 (B and C) shows photographs made in March, 1915, more than six years since operation.

Case II.—*Malunion of fracture of lower end of humerus; limited motion and cubitus varus.* William G., aged eighteen years. Treated in Dr. Harte's service at the Orthopædic Hospital. Referred by Dr. E. H. Kistler, of Lansford, Pa. When three years old this boy had fallen out of bed, landing on his left elbow. He recovered with Volkmann's contracture of the forearm, cubitus varus, and limited motion in the joint (40 to 110 degrees). When first seen, August, 1912, the Volkmann's contracture caused him no inconvenience, but the limited extension in the elbow was a serious handicap in his work in the mines, and his elbow was weak from the varus deformity and pained him if he used it much.

Photographs made before operation (Fig. 94) show the distortion of the bony points at the elbow and the limit of extension (× indicates head of radius; the condyles and the olecranon are indicated by dots).

*Operation* (September 2, 1912).—Through the usual external incision the head of the radius, which projected far backward (Fig. 95), was exposed posterior to the external lateral ligament. and was excised. The external condyle was then detached, the joint luxated, and a curved section was removed from the humerus, with Butcher's saw, much more bone being removed from the radial than from the ulnar side of the humerus, so as to overcome the varus deformity. Fig. 96 shows the portions of bone removed, that from the humerus having been removed in three sections, until the sawed surface fitted the ulna and the varus deformity was abolished. A flap of aponeurosis and muscle was secured from the triceps in the usual way. The epicondyle was reattached to the shaft of the humerus with chromic gut. A drainage tube was placed at each end of the incision. The tubes were removed after three days. It was not necessary to have employed them. The arm was dressed on a straight anterior splint, at an angle of 160 degrees.

September 5: Motion from 90 to 135 degrees is easy.

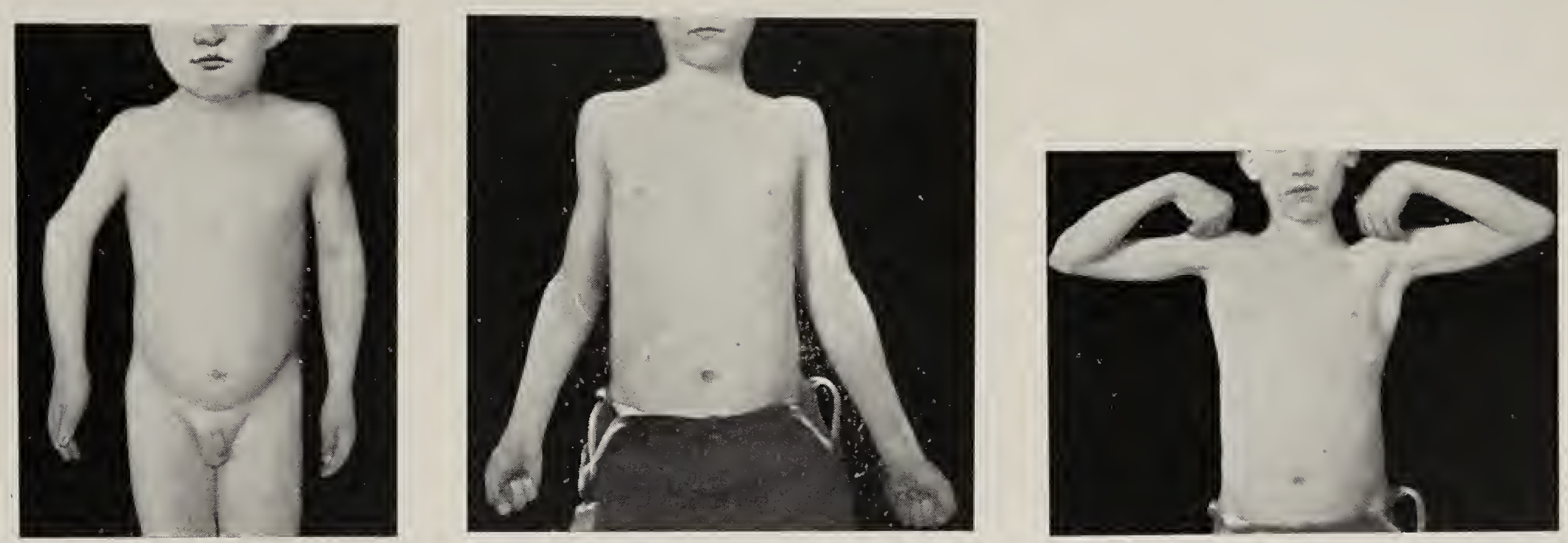
September 19: Out-patient. Sinuses (resulting from unnecessary use of drainage tubes) have healed. Motion 90 to 160 degrees is easy. He carries his arm in a sling.

October 3: Motion 65 to 135 degrees. Ordered massage and light passive movements three times weekly.

October 17: Treatment discontinued. Motion 65 to 160 degrees. Returns to work.

July 31, 1913: Eleven months after operation the patient was again photographed (Fig. 94), to show the range of motion (40 to 170 degrees). There was no cubitus varus and perfect function. The elbow is stable. He works on a breaker engine at the mines.

Case III.—*Bony ankylosis from metastatic arthritis.* Gertrude T., aged twenty-three years. Treated in Dr. Harte's service at the Orthopædic Hospital. In May, 1912, when about seven months pregnant, but without any evident cause (such as preceding tonsillitis, influenza, vaginitis, etc.) this patient developed an acute polyarthritis and was confined to bed for seven weeks. The pregnancy terminated normally after convalescence, but the left elbow and right knee were ankylosed. When first seen at the Orthopædic Hospital, in March 1913, about ten months after



*A* *B* *C*

FIG. 93.—Case I. *A* is a photograph taken in 1908, just before operation, showing cubitus varus. *B* and *C* are photographs taken in 1915, showing restoration of carrying angle and limits of extension and flexion.

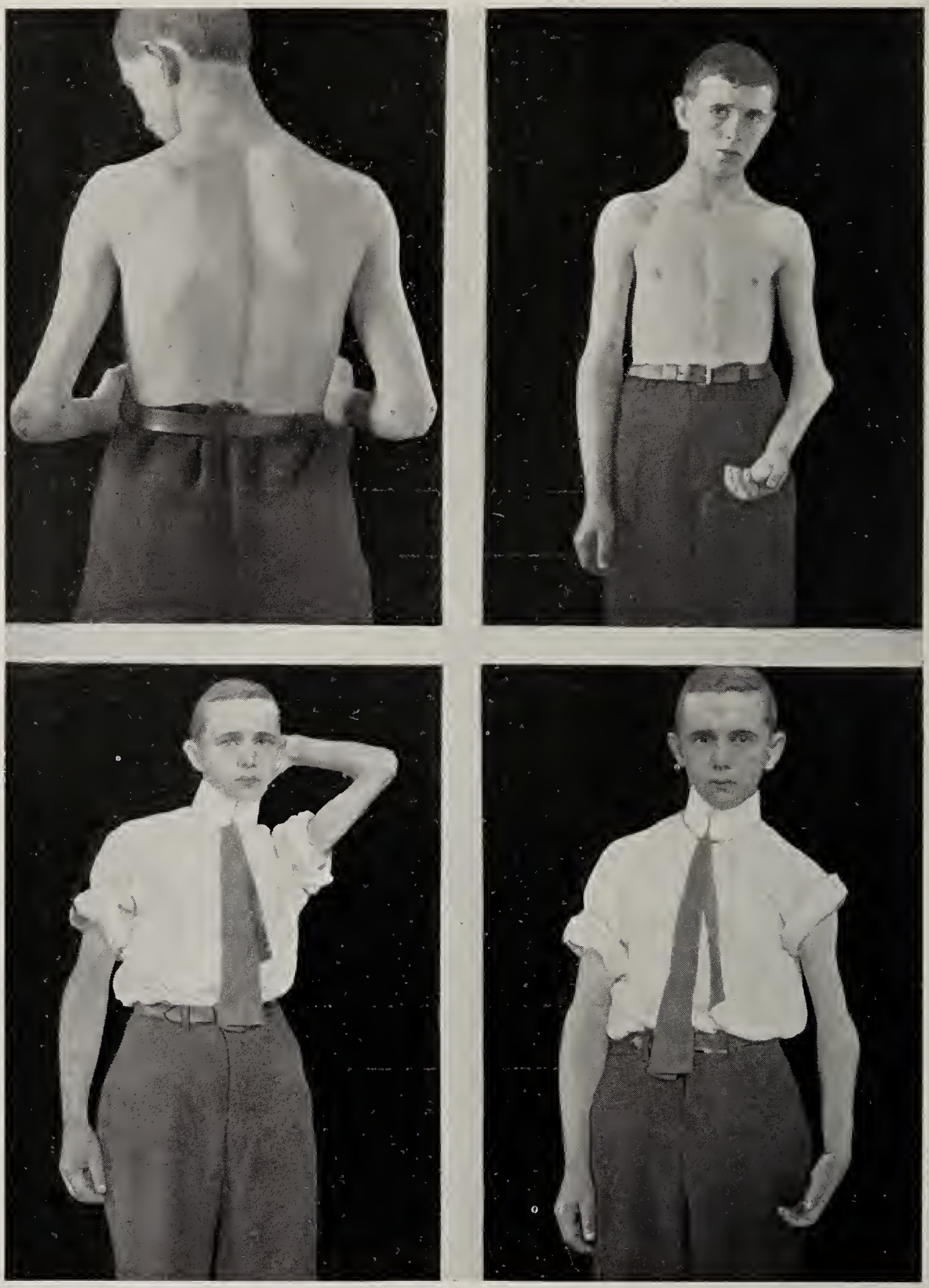


FIG. 94.—Case II. The first two photographs were made before operation, showing distortion of bony landmarks (× indicates head of radius) and limit of extension. The other two photographs were made eleven months after operation, showing range of flexion and extension, and restoration of carrying angle.

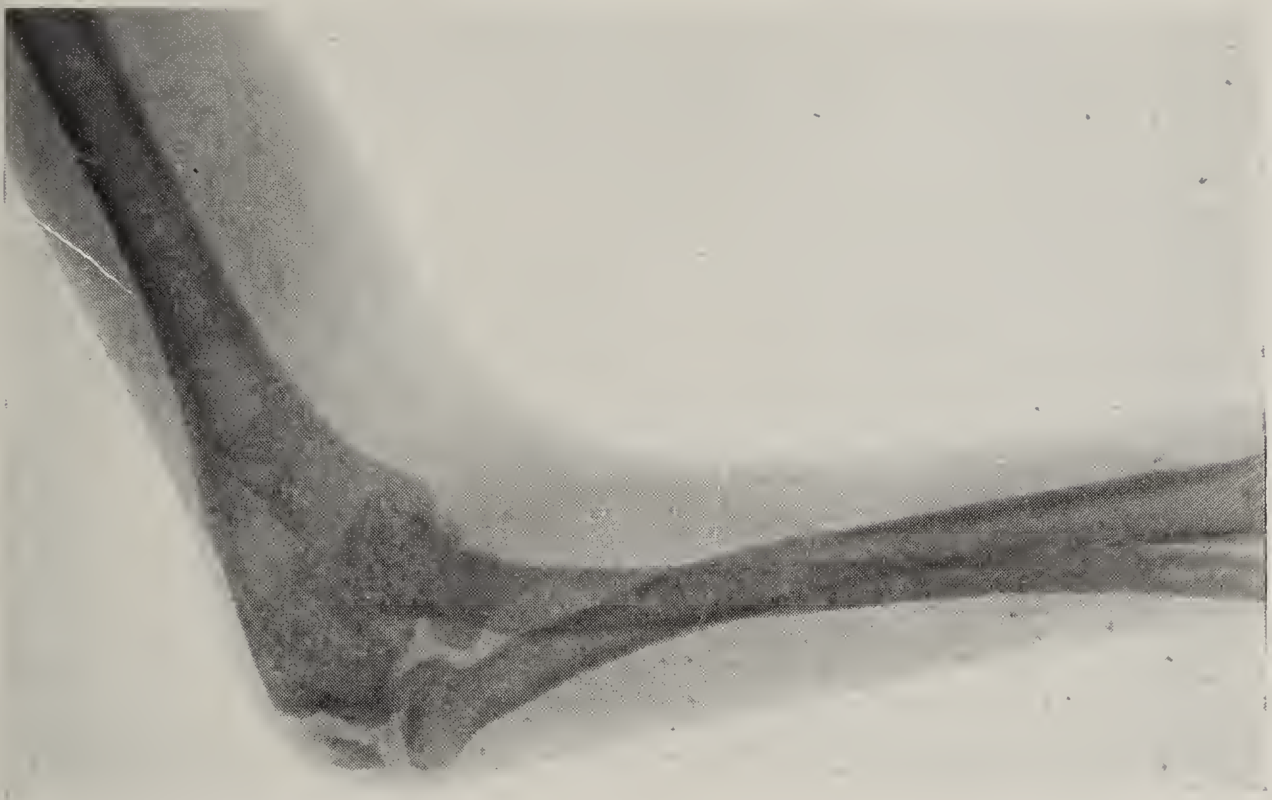


Fig. 95.—Case II. Skiagraph before operation, showing marked cubitus varus and limitation of extension.

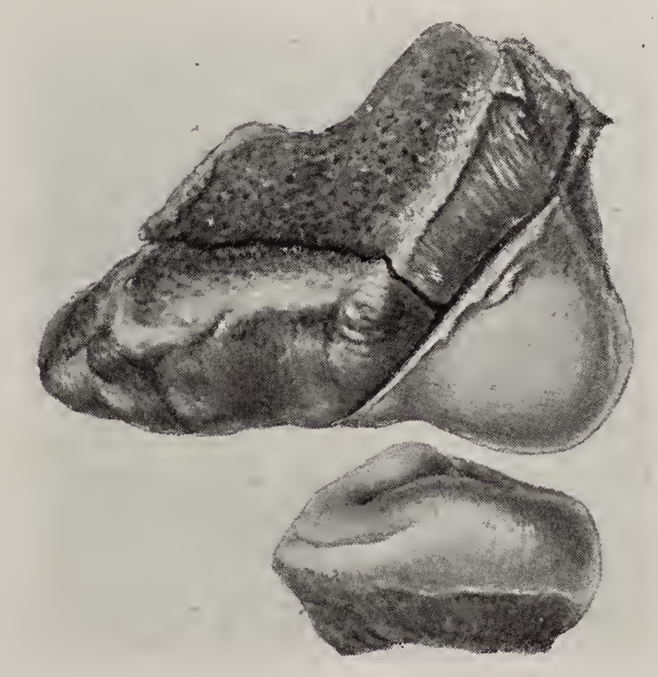


Fig. 96.—Case II. Arthroplasty of elbow; portions of humerus and head of radius excised (September, 1912).

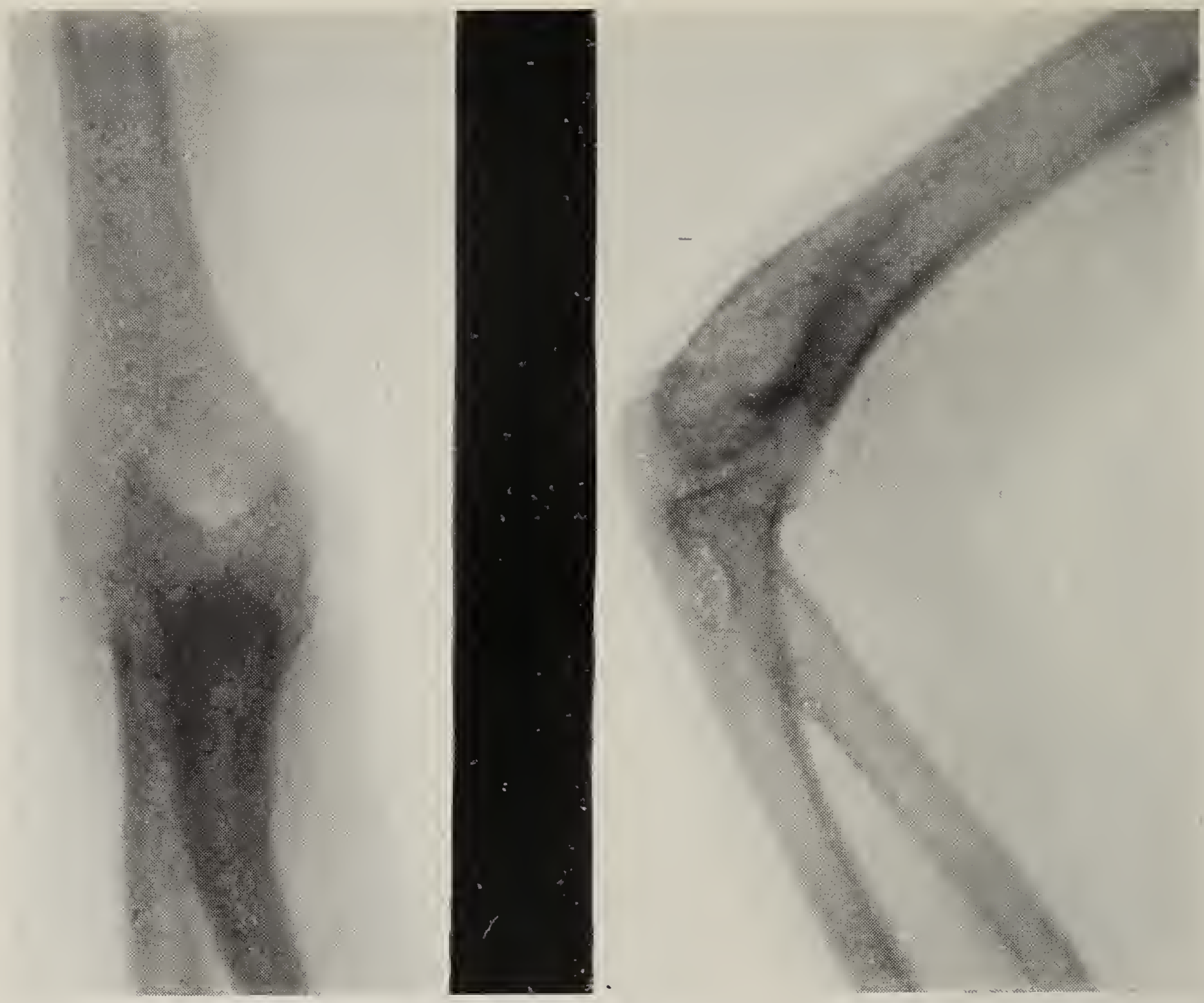


FIG. 97.—Case III. Skiagraphs showing ankylosis of elbow before arthroplasty.

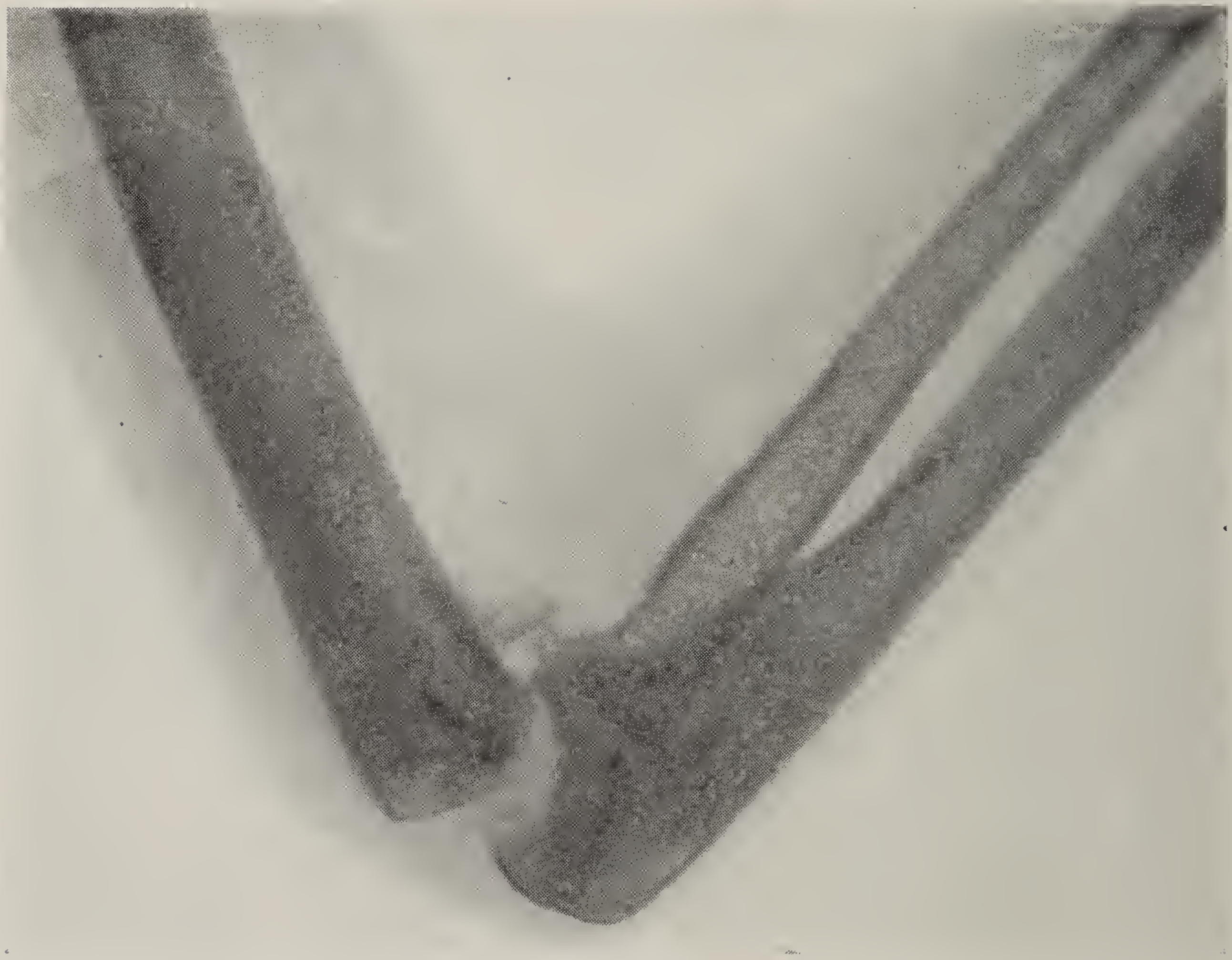


FIG. 98.—Case III. Result of arthroplasty. From skiagraph seven weeks after operation.

this attack of arthritis, the elbow was fixed in bony ankylosis at an angle of 110 degrees; fortunately the radiohumeral joint and the upper radio-ulnar joint were not involved, as rotation in the forearm was normal.

*Operation* (May 1, 1913). Arthroplasty of elbow by usual technique. A small incision was also made over the ulnar nerve and this was drawn away from the internal condyle until the bone ends were properly shaped. Flap obtained from triceps as usual, and epicondyle reattached to humerus by wire suture. No drain. Dressed on internal right-angled splint.

May 12: First dressing. Inner incision healed; outer incision healed all but one spot, between two sutures at upper end, over the cavity resulting from cutting the triceps flap. A little serous ooze occurred at this point. Motion of 30 degrees free and painless. Can get hand to mouth. Arm carried in sling.

May 15: Motion from 70 to 120 degrees without pain. Rotation in forearm normal. Can put hand to back of neck.

May 23: Passive motion from 65 to 160 degrees without pain. Active movement from 70 to 120 degrees. Arthroplasty of the knee was done today (*Trans. Coll. Phys. Phila.*, 1914, xxxvi, 236), and on this account the patient had to remain in the hospital longer. Fig. 73 shows the condition on admission, and Figs. 74 and 75 show respectively the limits of flexion and of extension in elbow and knee three months after operation. Figs. 97 and 98 are from skiagraphs made before and after arthroplasty of the elbow.

October 17, 1914: Eighteen months after operation there was motion in the elbow from 45 to 150 degrees, there was active power of extension in the triceps, and the joint was quite stable. She does all her own housework, and finds it a very useful arm.

CASE IV.—*Malunion of fracture of lower end of humerus, with limited motion.* Benjamin F., aged fourteen years. Treated in Dr. Ashhurst's Orthopedic Service at the Episcopal Hospital. In the summer of 1912 this boy fell on his elbow and sustained a fracture-dislocation of the type Posadas (diacondylar fracture of the humerus with forward displacement of the lower fragment and posterior dislocation of both bones of the forearm). Neither the fragments of the humerus nor the dislocation of the elbow had been reduced, and 16 months later the boy applied to the orthopedic department for disability from limited flexion and extension (65 to 150 degrees) (Fig. 99).

*Operation* (November 26, 1913).—Arthroplasty by the usual technique. After trimming the end of the humerus to proper shape with the bow-saw, it was found the ulna tended to stay in posterior luxation; so the olecranon was removed, preserving the periosteal attachments of the triceps. The radial head and the greater sigmoid cavity of the ulna were not disturbed. A flap from the triceps was inserted as usual, and the epicondyle was reattached by chromic gut sutures. No drain. Dressed on internal right-angled splint.

November 29: Discharged from ward. Skiagraph shows subluxation backward of radius and ulna.

December 1: Out-patient. First dressing; some sloughing of edges of skin flap turned back to expose triceps. Wound is clean. Elbow dressed in hyperflexion.

December 15: Skin granulating well. Free motion from hyperflexion to right angle. Arm in sling.

December 22: Motion 45 to 120 degrees. Out of sling.

January 5, 1914: Incision healed. Motion 40 to 145 degrees.

January 12: Motion 40 to 150 degrees.

January 19: Motion 40 to 160 degrees.

March 9: Motion 10 to 160 degrees.

April 27: Five months after operation (Fig. 99). Motion 10 to 180 degrees. In full extension radius and ulna luxate backward. There is free lateral motion in elbow, though external condyle is firmly attached to the humerus. Very slight power of extension in elbow, good power in flexion.

June 29: Seven months after operation. Can chop wood holding axe in both hands, indicating a considerable improvement in the stability of the elbow.

Case V.—*Bony ankylosis of elbow from septic arthritis.* Sarah M., aged twenty-seven years. Dr. Ashhurst's service at Episcopal Hospital (orthopedic). In December, 1913, this patient suffered from a "heavy cold" with cough; she was in the habit of carrying her eighteen-months-old baby on her left arm, which gradually became stiff and painful. In less than a week matter formed, and an abscess on the inner side of the joint was lanced by her family physician. She was admitted to Dr. Frazier's service in the Episcopal Hospital on January 8, 1914, and on January 20 Dr. Frazier opened the sinuses more freely and put in drainage tubes. Cultures of pus at this time gave a pure growth of Streptococcus pyogenes. The elbow became stiff. She went home in the end of February, and the sinuses were all healed early in March, 1914. Since then there has been no pain or tenderness.

Examination in May, 1914, showed ulnohumeral ankylosis at

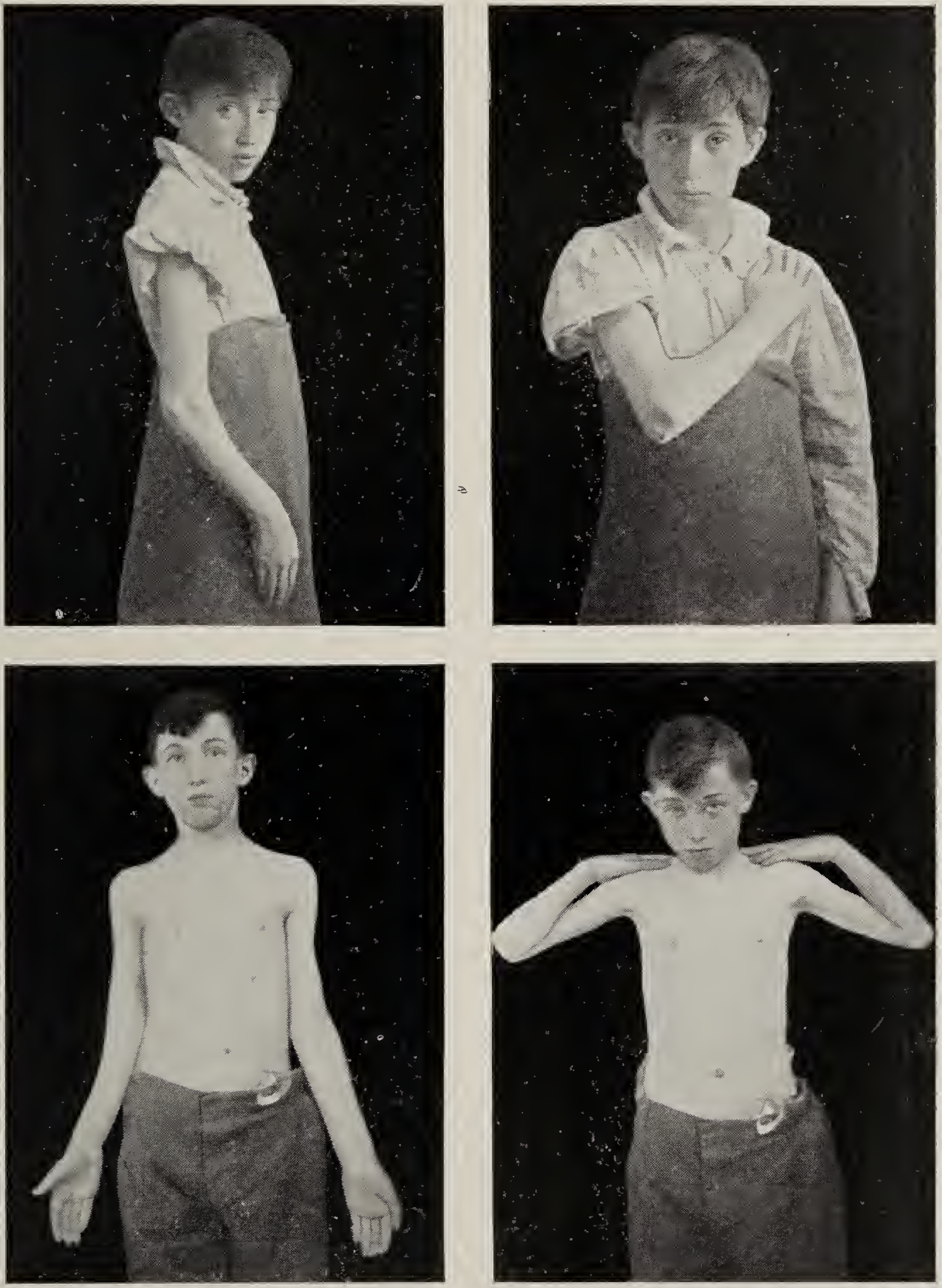


FIG. 99.—Case IV. Upper photographs show limits of extension and flexion before operation. Lower photographs were made five months after operation, showing results of arthroplasty.

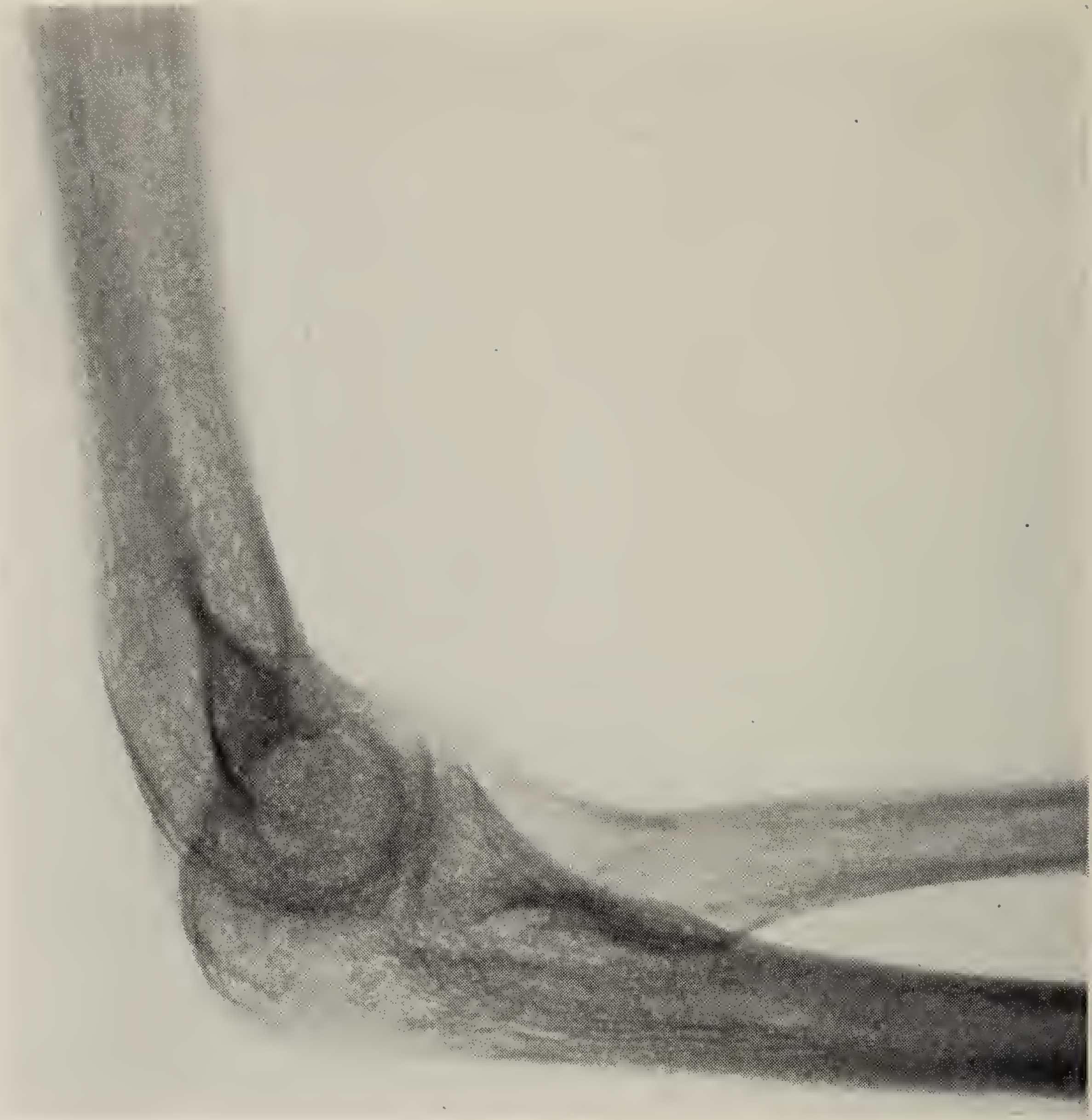


Fig. 100.—Case V. Skiagraph before operation, showing bony ankylosis.

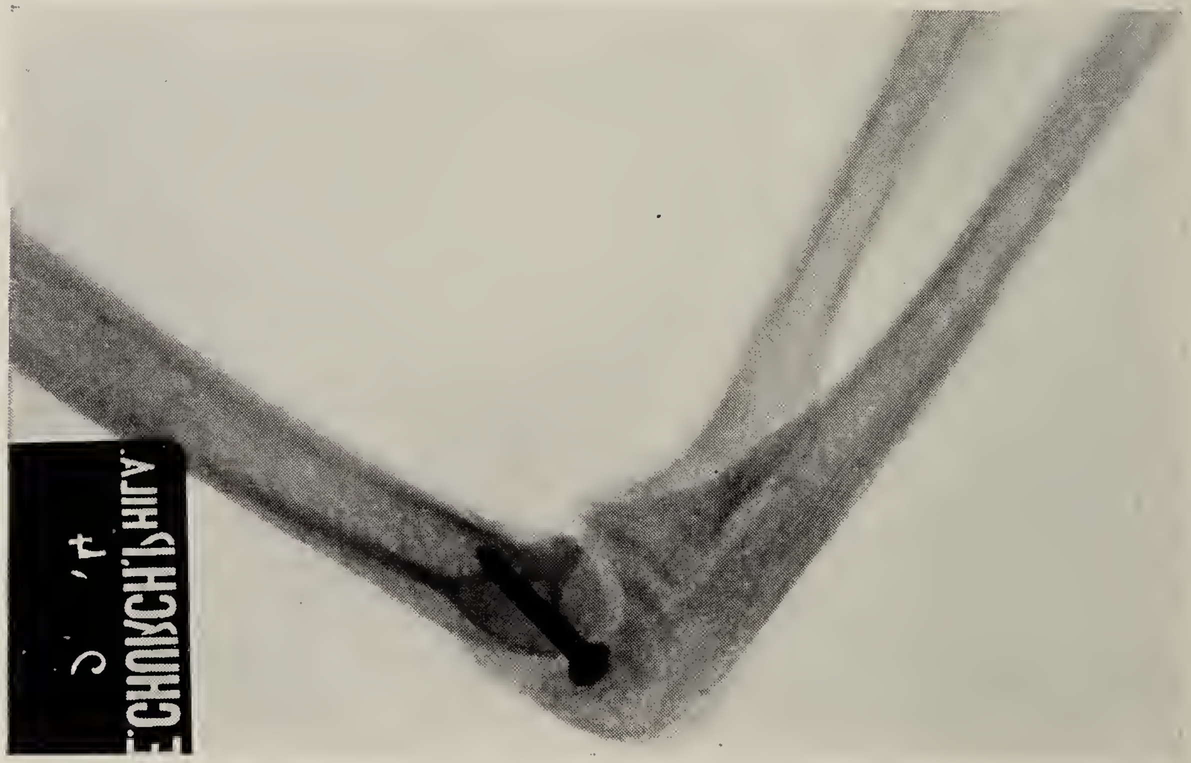


Fig. 101.—Case V. Skiagraph six weeks after arthroplasty.

an angle of 110 degrees. She could not get her hand to her mouth, nor even to the top of her head. Rotation in the forearm was about half-normal, supination being lost. Fig. 100 is from a skiagraph before operation.

*Operation* (May 30, 1914).—Arthroplasty by the usual technique. All told, sections about 0.5 cm. in thickness were removed from the humerus by the mounted Gigli saw. A flap from the triceps was interposed, and the external condyle reattached by a screw. No drain. Dressed on internal right-angled splint.

June 6: First dressing. Wound healed. Can put hand to face with ease.

June 8: Went home.

June 15: Out-patient. Motion 70 to 100 degrees. Massage ordered.

July 13: Motion 85 to 110 degrees causes pain at limits named.

August 31: Free and easy motion 90 to 120 degrees. Rotation normal. Refuses to have forceful motion under an anesthetic. Fig. 101 is from a skiagraph made six weeks after operation.

(NOTE: Examination more than two years after operation shows recurrence of bony ankylosis. Elbow is painless and hand can be put to mouth.)

SUMMARY OF RESULTS IN FIVE CASES OF ARTHROPLASTY OF ELBOW

| Case. | Before operation. | | | After operation. | | |
|---|---|---|---|---|---|---|
| | Flexion. | Extension. | Deformity. | Flexion. | Extension. | Deformity. |
| I | 40° | 145° | Varus | 35° | 150° | None |
| II | 40° | 110° | Varus | 40° | 170° | None |
| III | 110° | 110° | Ankylosis | 45° | 150° | None |
| IV | 65° | 150° | Posterior dislocation | 10° | 180° | Posterior dislocation (only in extension) |
| V | 110° | 110° | Ankylosis | 90° | 120° | None |

# CINEMATOPLASTIC AMPUTATIONS[1]

By ASTLEY PASTON COOPER ASHHURST, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

It does not appear that amputation for cinematic prosthesis, according to the method of Vanghetti,[2] has received adequate attention in this country. This emboldens me to report two cases in which I have resorted to this procedure, though the report is necessarily incomplete as it has not yet been possible to secure a satisfactory prosthesis, though the patients have good motion in their stumps.

The design of the operation, briefly, is to construct one or more muscular or tendinous loops at the end of the patient's stump, so that the voluntary movements of these loops may be transmitted to the artificial hand. Many artificial arms are already on the market for use with ordinary stumps. In the case of one of those in most general use, the hand is opened by touching a spring with the other hand, and snaps shut again, into a fist, when the spring is released. Such a hand as this, as the manufacturers themselves are forced to admit, is useful for nothing more than "Sunday wear," as there is no voluntary grasp in the hand. A better type of artificial hand is one that secures its motion chiefly by means of straps passed around the patient's body, especially over the opposite shoulder. With one type of arm constructed on this principle, the patient is enabled, presumably only after long and constant practice, to perform almost any motion; and a patient with both arms amputated can dress himself, feed himself, and can make many graceful gyratory motions of little practical use. At the recent meeting of the International Surgical Society in New York City, opportunity was afforded to see a number of patients equipped with arms of this type; but on closer examination I found that while almost any motion was possible, with fingers, thumb, wrist and elbow, yet the grip was very weak (unless the hand was locked by a spring), so that no manual work was attempted. These men make their living advertising this particular make of artificial arm, as travelling salesmen; and probably could make as good a living without any artificial arm if they advertised some other article of merchandise.

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[1] Read before the Philadelphia Academy of Surgery, May 4, 1914. Reprinted from Annals of Surgery, 1914, ii, 751.

[2] G. Vanghetti: Plastica e Protesi Cinematiche; Nuova Teoria sulle Amputazione e sulla Protesi. Empoli, 1906. Previous works by this author, on the same subject, had been published in 1898, 1899, and 1900.

But there are very many patients in the laboring classes who are unable to live by their brains, and are utterly incapacitated by the loss of an arm; the most they can do is to act as watchmen, gate-keepers, elevator men, etc. If, however, they could be provided with a hand movable at will and possessing at the same time a grip strong enough to wield

"a shovel, a rake, or a hoe,
a pickaxe or a bill"

or do other laboring, carpentering, masonry or painting work, their earning capacity would be considerably increased, even if it still fell short of the normal.

The amputation for cinematic prosthesis is a tedious operation and is not designed as a primary procedure in traumatic cases or in other patients acutely ill. The primary amputation in such cases should be given ample time to heal before the cinematoplastic amputation is undertaken. The level of bone section is determined by the length of the soft parts available for making the muscular loops; the latter should be amply long, so as to allow for subsequent retraction and nevertheless provide for plenty of play beyond the bone end.

In both the cases in which I have adopted this method, the amputation was done through the humerus, by the following technique (Figs. 102 to 105), which differs somewhat from that described by Vanghetti: a small skin flap is outlined over the brachial vessels, as long as the diameter of the limb and nearly an inch wide, with its base at the level proposed for section of the bone (Fig. 102, *AB*); this flap is raised with the sub-

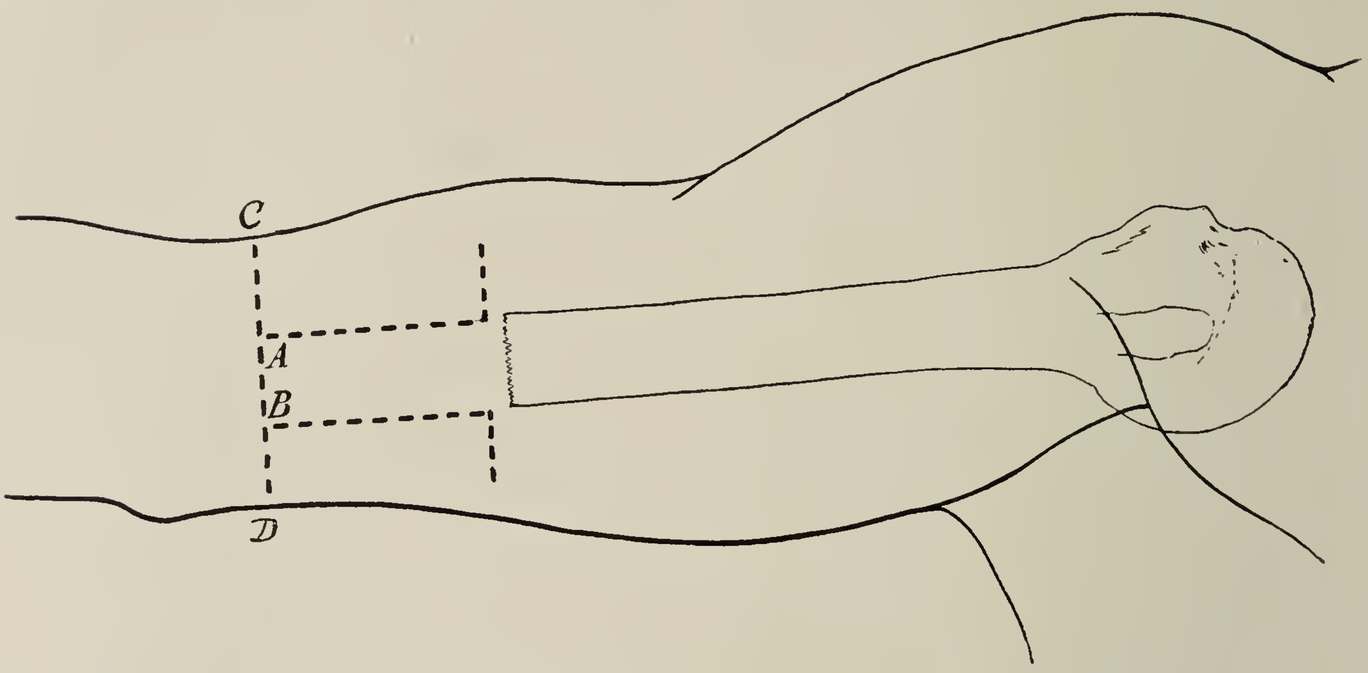

FIG. 102.—Cinematoplastic amputation. Inner surface of arm; the flap, *AB*, is to be used to cover the end of the bone; circular amputation at *CD*.

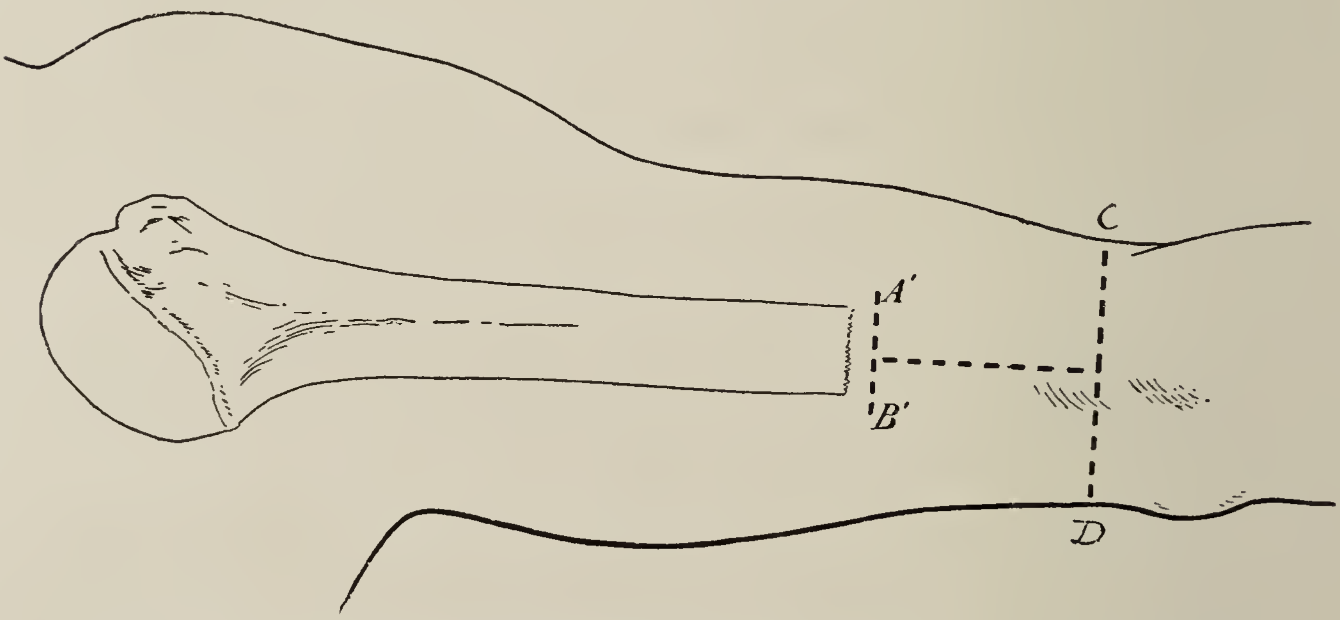

FIG. 103.—Cinematoplastic amputation. Outer surface of arm; the flap, *AB*, is sutured to the line *A′B′*.

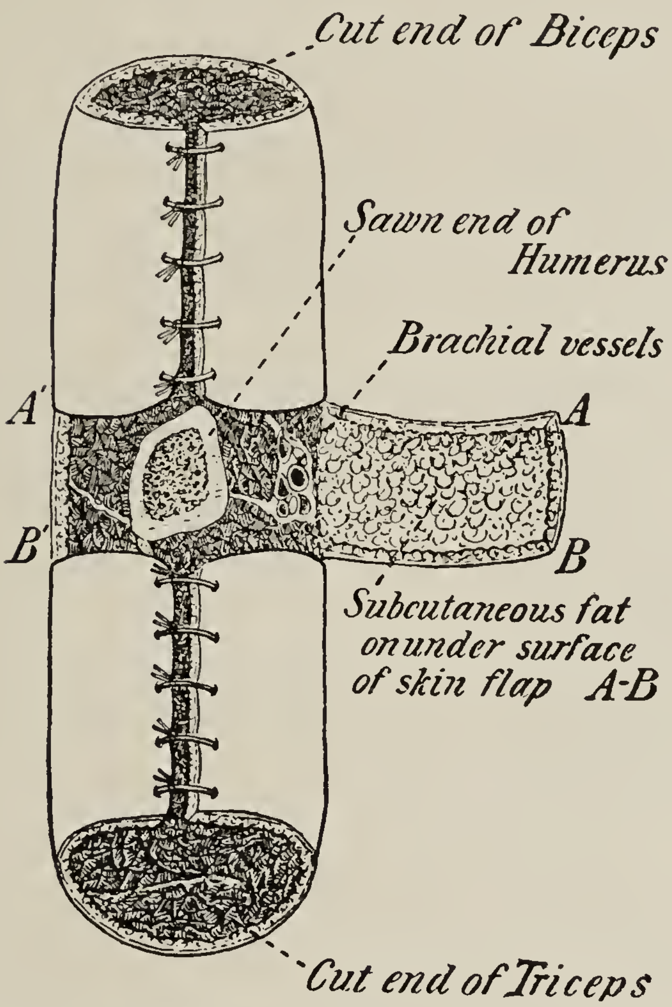

FIG. 104.—Cinematoplastic amputation. Diagrammatic view of the end of the stump. The flap, *AB*, is sutured to *A′B′*, and the skin overlying the muscular flaps is sutured around them as a cylinder.

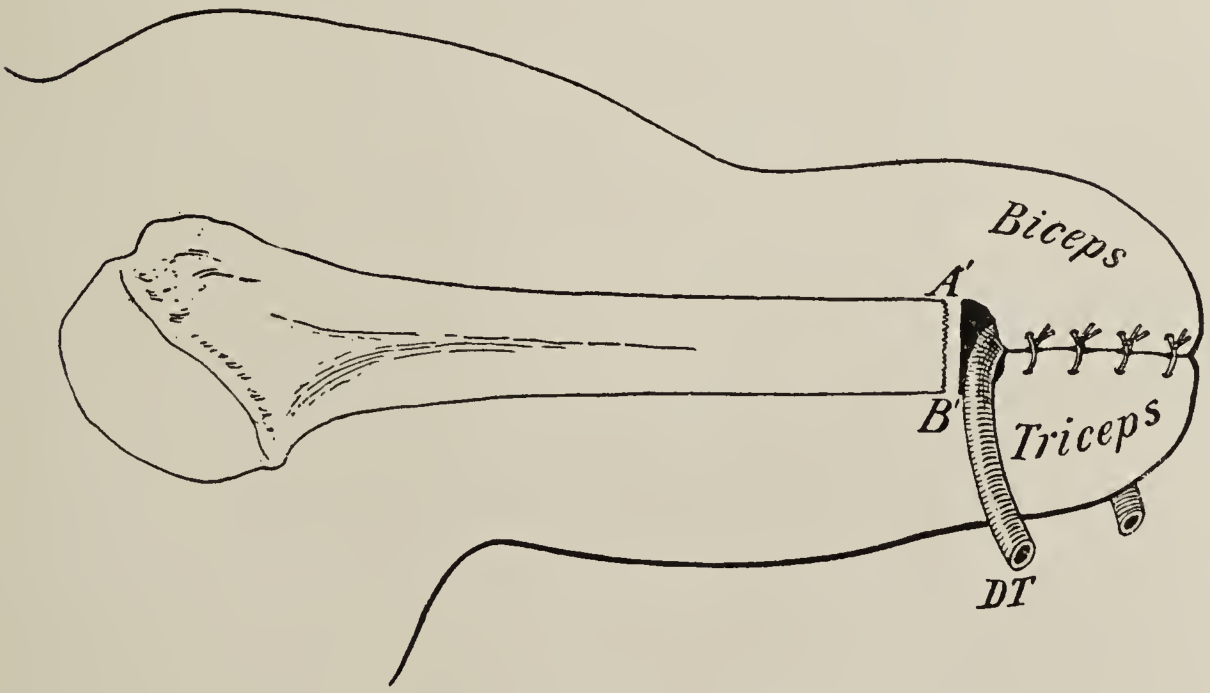

FIG. 105.—Cinematoplastic amputation. The biceps has been sutured to the triceps, and a rubber tube is passed through the loop before dressing the stump.

cutaneous tissues, and the brachial artery and vein are ligated and divided just above the level at which the bone is to be sawed. The nerves are divided at the same level or higher, but great care is exercised throughout the operation not to interfere with the nerve supply of the muscles which it is proposed to utilize in the stump. A longitudinal incision is then made on the outer side of the arm (Fig. 2), between the flexor and extensor muscles, and these with the overlying skin are then raised from the bone, from the level of proposed bone section down as far as possible. In the arm the musculospiral nerve is now divided, unless it was accessible from the first incision. The soft parts are then divided circularly down to the bone at the distal limit of healthy tissues, and the musculocutaneous flaps are raised; the anterior flap contains the biceps (perhaps also some of the deltoid, brachialis anticus, or coracobrachialis, according to the level), and the posterior flap contains the triceps. The bone is then sawed at the desired level. A small transverse incision is then made toward the centre of the flexor and extensor flaps through the skin only, at their bases (Fig. 102), so as to permit of wrapping the skin around the biceps and triceps respectively, in the form of a cylinder. This little procedure when repeated on the outer side of the stump also leaves a free skin margin (Fig. 103, *A′B′*) to which may be sutured the end of the skin flap designed to cover the bone. This flap is next adjusted across the end of the bone, and is sutured in place with chromic catgut (Fig. 104, *AB* is sutured to *A′B′*). Absolute hemostasis is important. Then the skin overlying the muscular flaps is wrapped around them in a cylinder, so far as possible (the skin usually is too scanty, and I had to sacrifice some of the muscle in both cases), and is sutured. Next, the free ends of the biceps and triceps are sutured to each other, end on, with buried sutures of chromic gut, and the remainder of the skin is finally closed as accurately as possible. A large rubber tube is passed through the loop thus constructed (Fig. 105), and the stump is lightly dressed.

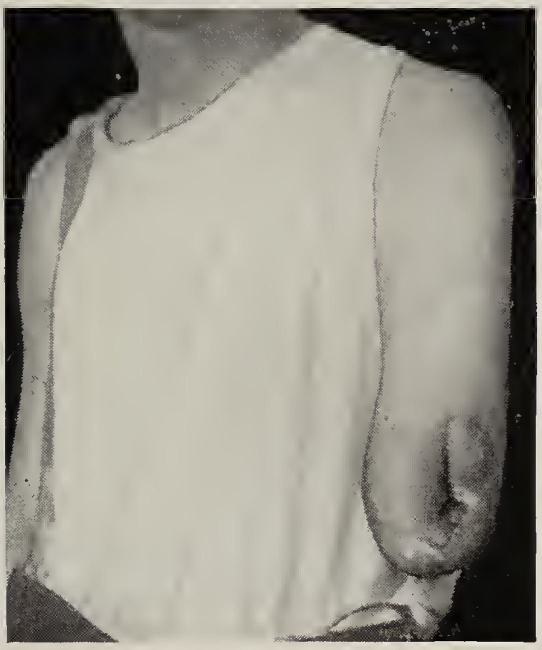

Fig. 106.—Cinematoplastic amputation; first patient; four weeks after operation.

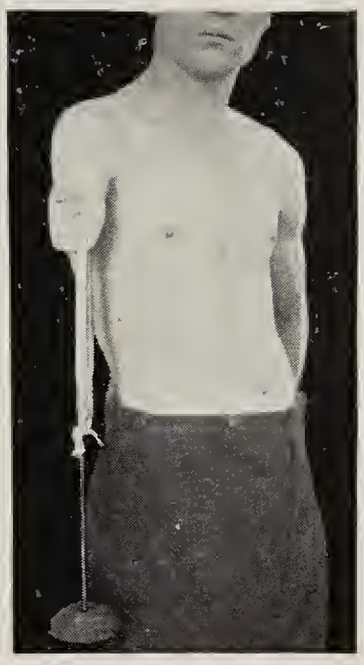

Fig. 107.—Cinematoplastic amputation; second patient; five months after operation.

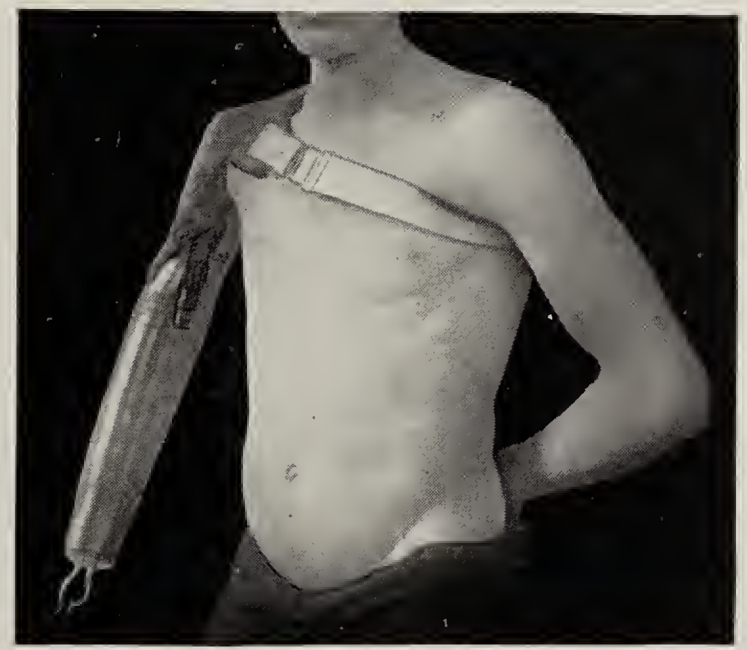

Fig. 108.—Cinematoplastic amputation; second patient; temporary prosthesis.

Case I.—Man aged thirty-five, a steam-fitter by occupation, was in Dr. Frazier's service at the Episcopal Hospital. Amputation was done August 14, 1911, at the middle of the humerus for incurable infection of the hand and forearm, of nearly four months' duration. In this case the cinematoplastic amputation was done as the primary procedure. There was prolonged but not very active suppuration, due chiefly to an intractable dermatitis of the stump, which did not heal permanently until four months after operation. Fig. 106 shows his condition four weeks after operation.

As soon as healing was complete he left the city, and I did not see him for almost two years. In November, 1913, he returned to Philadelphia and I found his stump in very good condition. The flaps had retracted considerably, and the loop is now about one-third smaller than shown in the photograph taken four weeks after operation. His stump is strong and freely movable voluntarily over the end of the humerus, and there is a direct pull of at least half an inch (1.25 cm.). He has surprising strength in the muscular loop, and if fitted with a proper prosthesis should have a strong grip in the artificial hand.

Case II.—Young man of twenty-one years, a painter by trade, whose arm had been amputated first for a crush at the age of fourteen years, at about the middle of the humerus. When nineteen years of age (in 1910) reamputation had been done for a conical stump, the bone being divided this time through the insertion of the deltoid. Though this left a very short stump, it seemed that a cinematoplastic amputation should increase its usefulness.

The operation was done in Dr. Frazier's service at the Episcopal Hospital on October 18, 1912. Healing was prompt and good power was secured in the stump, with nearly one inch of direct pull. The patient himself claims at least an inch and a quarter (3 cm.), but I think this is an exaggeration. Fig. 107 is from a photograph made five months after operation.

This patient also was lost sight of for a long time, and only recently has he returned for observation. He has spent some time in jail, has neglected his stump, and thought the loop had closed up; but a strip of gauze was easily drawn through, though it caused a little bleeding, and a very slight discharge persisted as long as the gauze was kept in place. The stump is very strong, however, and the loop can be both flexed and extended very actively.

I myself and the patients themselves have consulted several manufacturers of artificial arms, with the object of having a cinematic prosthesis applied. The manufacturers invariably recommend and prefer artificial arms of their own manufacture and decline to manifest any enthusiasm about making artificial arms with a different mechanism. Finally I induced one manufacturer to make the attempt, but the patient then in question declined to pay for any "experimental" arm unless it proved absolutely satisfactory. This guarantee the maker declined to give, and the proposed deal fell through. This patient (Case I) then conceived the idea of making his fortune by inventing an artificial hand for himself, having it patented, then inducing some manufacturer to put it on the market and pay the royalties to him, while he himself would enjoy life travelling over the world as a sales agent. Though he assured me he would send all the patients with arm stumps to me to have a cinematoplastic amputation performed, I have heard nothing further from him, and fear his scheme is not working out as well as he had anticipated.

Finally I determined to have a very simple apparatus constructed for the second patent, who was more tractable, and appeared to be willing to become the subject of experimentation. In default of any better contrivance, and purely as an experiment, I instructed Mr. Henry Saur of this city to make a pair of fingers, or finger and thumb in the form of pincers, attached to an arm socket, and worked on the principle of levers, by means of a cord which passes through the loop in the patient's stump. If this proves practicable, it can be extended to include more fingers or a complete artificial hand (Fig. 108).

I may add that Vanghetti himself experienced very great difficulty in having a suitable prosthesis constructed, and the fact that he succeeded at last probably is due to his having as great a genius for mechanics as for surgery. However this may be, the various designs of apparatus which he gives in his book have not served to inspire any useful ideas in the mechanicians to whom I have submitted them. Hence I make this report of these amputations for cinematic prosthesis merely to show that the construction of the cinematoplastic stump is quite possible, and in the hope that someone with more mechanical ability than I myself possess will undertake the further problem of designing the prosthesis.

# THE USE OF SILK LIGAMENTS AND TENODESIS IN INFANTILE PARALYSIS[1]

By EDWIN P. LONGAKER, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

Recently it has been my good fortune to have observed a few cases of infantile paralysis in which the application of silk ligaments alone or combined with other measures seemed indicated.

To Dr. Ashhurst, in whose service these patients were treated, I am indebted for the privilege of reporting these cases.

Case I.—Paralytic varus, right foot; paralytic valgus, left foot.

Mildred S., aged six years; admitted March 3, 1914.

*Family History.* Father and mother living and well.

*Personal History.* Negative.

*History of Present Illness.* At three years of age she had an attack of paralysis and has been wearing braces for some time, following a long course of massage and electricity.

Examination shows the following: Right lower extremity, foot in slight varus; paralysis of all the muscles of the leg excepting very slight power in the iliopsoas, biceps, and the flexors of the toes, and doubtful action in the posterior tibial and calf muscles. Left lower extremity, deformity is valgus and planus and paralysis of the anterior and posterior tibials.

*Operation* by Dr. Ashhurst, April 4, 1914. Right leg, silk ligaments to overcome varus; left leg, lengthening of tendo Achilles and Putti's operation.

Right leg, under Esmarch anemia; flap incision along the tibia 2½ inches above the ankle, periosteum separated for a distance of one inch and a hole bored across the tibia, 2½ inches above the ankle; two small incisions were made below the ankle and a hole bored through the tarsus from one incision to the other. Silk ligament was run through the hole in the tibia and carried down under the anterior annular ligament and through the holes in the tarsus. The two ends were tied above the ankle, with the foot pulled up into dorsiflexion. The foot still remained in slight varus, so another silk ligament was run through holes drilled in the lower end of the fibula and through the cuboid; this, when tightened, served to bring the foot into good position. Skin closed with No. 1 chromic gut and the foot and leg put in plaster cast.

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[1] Read at a meeting of the Episcopal Hospital Clinical Society, May 18, 1914.

Left leg, under Esmarch anemia. The tendo Achillis was lengthened by the usual Z technic; the tibia was exposed above the annular ligament and was drilled transversely; the tendon of the tibalis anticus was cut from its muscular origin and was split for an inch or more. One of the split ends was then drawn through the hole drilled in the tibia until the foot was pulled up into an overcorrected position (slight calcaneovarus); the two split ends of the tibialis anticus tendon were then sutured to each other and to the periosteum of the tibia, thus anchoring the tibialis anticus to the bone above the annular ligament, and converting it into a ligament. The skin wounds were closed as usual and a plaster cast applied.

May 27, 1914. Gypsum cases removed. The silk ligament has ulcerated through on the flexure of the ankle, and is exposed for 2 inches.

May 31. The anterior silk ligament is removed.

June 20. The external silk ligament is removed through a sinus.

June 22. Sinuses all healed.

July 21. Went home, walking in braces, with a little support to hands.

September 21. Both feet in good position, but in walking (which she can now do alone), the right foot has tendency to varus and the left to valgus.

February 26, 1916. Nearly two years after operation the father writes that the girl walks very well; no longer needing braces.

Case II.—John J., aged six years; admitted April 6, 1914.

*Family History.* Negative.

*Personal History.* Negative except for an appendicular abscess in 1912 which was operated on and took six months to heal.

*History of Present Illness.* In August, 1909, when twenty-six months old, he had an attack of infantile paralysis in both legs and right arm. He went to the Orthopædic Hospital in 1910, where he had massage and electricity. Brace ordered in November, 1910.

Examination on admission to the Episcopal Hospital, shows the following: Right leg: Foot-drop and slight weakness of internal rotators of hip; paralysis of extensors of toes, and extensor longus hallucis and tibialis anticus; weakness of the calf muscles; peronei and flexors of the toes fairly good. Valgus is present. Left leg: Weakness of internal rotators and of calf muscles. Peroneals and posterior tibia tend to luxate in front of malleoli; there is inclination to calcaneus and cavus.

*Operation* by Dr. Ashhurst, April 8, 1914. Tenodesis and silk ligament. In right foot under Esmarch anemia, a flap incision was made along the tibia extending up from the ankle 3½ inches. Periosteum of tibia stripped off and a longitudinal groove made in the bone; next, a hole was bored through the tibia at the level of upper end of the incision, through which hole the silk ligament was pulled; two small incisions were made below the ankle and a hole bored through the tarsus, probably piercing the scaphoid and cuboid; the silk was then pulled through this and the two ends tied above the ankle, the foot thus being brought into good position. The tendon of the tibialis anticus was pulled taut, drawing the foot into slight varus position, and was placed in the groove in the tibia along with the inner cord of the silk ligament and sewed down under the periosteum. Incisions closed with No. 1 chromic gut and the leg put in a plaster cast.

May 25, 1914. Gypsum case removed. Foot in admirable position, and tendon of tibialis anticus feels taut under the skin. No tendency to varus. Brace applied.

November 9, 1914. Foot-drop has recurred. Quite an effusion and thickening where tibialis anticus was buried in tibia. Slight tendency to varus.

March, 1916. Marked valgus on right; calcaneovalgus on left. Suitable case for arthrodesis and horizontal section.

CASE III.—Leonard C., aged seven years, admitted April 4, 1914.

*Family History.* Negative.

*Personal History.* Negative.

*History of Present Illness.* Patient had an attack of paralysis at five years, otherwise he has been well. He went to the Orthopædic Hospital in 1909; had massage for two years, then wore braces for two more years, and for one year has had no support. Operation was advised one year ago. In June, 1913, he had an operation done on the lumbar spine at another hospital, with no improvement. (Presumably an attempt to secure return of function by anastomosis of filaments of the cauda equina).

Examination on admission to the Episcopal Hospital shows the following: The patient walks with a marked limp in the left leg. He has to put the hand on the thigh at each step to prevent the knee from collapsing. The limb is in external rotation, the foot in valgus, and some cavus, no contractures are present. The left lower extremity shows very slight power in the iliopsoas, fair power in the tensor fasciæ femoris, none in the gluteus medius, slight in the gluteus maximus; none in the adductors or hamstrings; slight power in the quadriceps, but not sufficient to keep the leg extended on the thigh. Paralysis of anterior and posterior tibials and flexors of the toes. The extensor longus digitorum overacts, and there is very slight power in the calf muscles.

*Operation* by Dr. Ashhurst April 22, 1914. Tenodesis and silk ligament for paralytic foot-drop and valgus of left leg.

Under Esmarch anemia, flap incision along tibia, periosteum stripped back for 3 inches and a longitudinal groove made in the bone, in which the tendons of the tibialis anticus and extensor longus digitorum were included and covered with periosteum, sewed down with No. 1 chromic gut, while the foot was held at right angles with the leg. This failed to overcome the valgus, therefore a silk ligament was run from the tibia to the scaphoid, but not under the annular ligament in this case. The two ends were tied above the ankle, after the valgus had been overcome by pulling the ligament tight. Incisions closed and leg put in plaster cast. The external rotation of the thigh was overcome by transplanting the tensor fasciæ femoris into the great trochanter.

November 2, 1914. Walks well with brace.

February 8, 1915. Valgus has recurred.

February 20, 1915. Subastragalar arthrodesis done, the boy now being eight years old, and skiagraphs showing enough bony development for this type of operation.

October 29, 1915. Foot firm and in excellent position. Has worn no brace for several months. Recently a sinus has formed above the internal malleolus, and the silk ligament inserted 18 months ago has begun to work out. A strand of silk six inches or more long is pulled out. Sinus closed in a few days.

Notes of the following additional cases in which tendon fixation or silk ligaments were employed may be of interest:

Case IV.—Joseph di N., aged seven years. Valgus, right.

May 24, 1913. Operation by Dr. Ashhurst at the Orthopædic Hospital. The peroneus longus was transplanted to the base of the first metatarsal, and a silk ligament was attached to the tibia and the scaphoid, drawing the foot into slight varus.

July 8, 1913. Cast removed, and brace applied.

March 10, 1914. The silk ligament has not prevented recurrence of the deformity, but has caused no irritation.

December, 1915. Transverse horizontal section of tarsus was done by Dr. G. G. Davis, for valgus with tendency to calcaneus.

Case V.—Louis I., aged six years. Varus and foot-drop.

April 23, 1914. Operation by Dr. Ashhurst at Episcopal Hospital. Tibialis anticus and extensor longus digitorum fixed in groove in tibia, but this did not overcome varus; therefore proximal end of peroneus longus was transplanted in front of external malleolus and fixed in groove in tibia.

June 4, 1914. Brace applied.

August 22, 1914. All tendons have pulled loose, with recurrence of foot-drop and varus, but not so bad as before operation.

April 10, 1915. Second operation by Dr. Ashhurst at Orthopædic Hospital. The undivided peroneus brevis was shifted in front of external malleolus and fixed in a groove in tibia. The peroneus longus (previously transplanted to this situation) was found adherent to the periosteum but not sufficiently taut; it was reimplanted in a new groove. The tibialis anticus was found sliding freely in a sheath; apparently the sheath had not been excised at first operation; the tendon was again buried in a groove in the tibia.

September 4, 1915. Tendons have pulled loose, and foot drops to angle of 135 degrees with leg. Arthrodesis advised for valgus which has developed since operation.

Case VI.—Matilda K., aged five years. Equinovarus, left.

May 20, 1914. Operation by Dr. Ashhurst at Episcopal Hospital. Lengthening of tendo Achillis and silk ligament from tibia to cuboid and scaphoid. Kept in ward seven months, owing to nearly complete paralysis of both lower extremities.

December 28, 1914. Can walk some by pushing stool in front of her.

December 2, 1915. Foot stays in fairly good position; the silk ligament appears to have held.

Case VII.—Sadie Z., aged five years. Equinovalgus, left.

April 10, 1915. Operation by Dr. Ashhurst at Orthopædic Hospital. Lengthening of tendo Achillis, transplantation of extensor longus hallucis to scaphoid, and fixation of tibialis anticus in tibia.

February, 1916. Valgus has recurred, but foot more stable than before operation.

The silk used in these cases was No. 6 twisted, prepared in the following manner: boiled in bichloride of mercury 1 to 1000 for one-half hour, dried, and the next day (*i. e.*, the day of the operation) boiled in paraffin for one-half hour.

In these cases all casts should remain on at least twelve weeks; this is done because surgeons have found that the silk breaks or tears if the casts are removed too soon. The patients operated upon were all under eight years of age, and the *x*-ray picture showed that the bones in the ankle were not fully developed, therefore arthrodesis was contra-indicated.

Lange, in 1898, was the first to use silk in surgery of the tendons. In 1903 he first used it as a ligament in the joints. Many European surgeons have used silk in infantile paralysis with fair success. Bartow, Plummer, and Allison in this country report good results from its use. The two former, Bartow and Plummer (*Am. Jour. Orthop. Surg.*, 1911) have used it in over 60 cases, and they have had no trouble from sloughing, and they claim there is less mutilation of the joint than in arthrodesis. In many cases braces can be dispensed with, though it is probably better to use them for a little while after removing the casts as will be done in ours. At a later date arthrodesis can be performed if deemed necessary. Nathaniel Allison (*Am. Jour. Orthop. Surg.*, May, 1913), after animal experimentation, says: "It seems that the silk implant, which has been properly prepared before it is placed in the tissues, causes a reaction which is that of an attempted organization of the foreign substance. There is no effort on the part of the tissues to rid themselves of the silk, but rather an attempt to produce fibrous bands, using the silk as a trellis upon which the proliferation may take place. It may be assumed that, provided a sterile operation is done, and provided the silk is properly prepared, it will remain indefinitely in the tissues. Its ultimate fate remains to be determined."

In our cases, the silk was reinforced by a *tenodesis*, or fixing the tendons in the bone. It is often advisable to make suitable combinations, whereby the silk is strengthened and less apt to break.

By fixing in bone the proximal end of the paralyzed tendons, they are converted into ligaments, as their distal ends are already attached to bone. Mere shortening of the tendons does not accomplish the same result, as the muscular part will soon stretch, unless this is entirely eliminated by attaching the proximal ends of the tendons (where they arise from muscular fibres) to bone. The tendons themselves will not stretch. Two methods are used for attaching the tendons to bone. In Putti's method a hole is drilled in the bone and the tendon (or a portion of it after it has been split) is drawn through this hole; in Codivilla's method, which is simpler, a longitudinal groove is cut in the bone and the tendon is buried in this by suturing the periosteum over it. Neither of these methods should be employed in the case of the tendon of any muscle in which recovery of voluntary motion might possibly occur; they should be used only for tendons arising from muscles which are hopelessly paralyzed, as the operation throws the muscle out of commission entirely, and it would be difficult to undo tenodesis subsequently, or to reunite the proximal end of the tendon to its muscle should the latter ever recover its function.

It is too early to draw any very definite conclusions in these cases, yet it seems likely that the operation will have an important place in the many methods for the fixation of paralytic joints. Not only can it be applied at the ankle but it has been used in other joints.

In concluding, I would say that the use of silk ligament alone or with tenodesis is especially applicable in those cases of infantile paralysis, which are too young for arthrodesis and which have no tendons that can be transplanted, and in which good position, stability, and prevention of deformity are desired and in which the indefinite continuation of support by external apparatus is objectionable.

# SARCOMA OF THE CLAVICLE AND THE SCAPULA

By A. BRUCE GILL, M.D.

ASSISTANT IN THE ORTHOPEDIC DEPARTMENT OF THE HOSPITAL

Dr. Bruce Gill reported the following case which occurred in the orthopedic service of Dr. Ashhurst at the Episcopal Hospital.

A woman aged fifty-one years presented herself complaining of a lump on her left shoulder and of pain in the shoulder, particularly on motion. The mass had first appeared about 4 weeks before her coming to the clinic and had gradually increased in size. For 2 years prior to her admission she had been having pain in her left shoulder on motion of the arm. This pain was sharp, paroxysmal, and was referred down the arm. It was relieved by heat and increased by massage and motion. The patient could ascribe no cause for the pain and the appearance of the tumor and stated that she had never suffered any injury to the arm or shoulder.

She was admitted to the Hospital on July 28, 1914. She was pale and poorly nourished and had been losing weight. On the top of her left shoulder in the neighborhood of the acromioclavicular joint was a mass about 1½ inches in diameter, rounded, soft, fluctuating, slightly red and warm, and slightly tender. It was not painful except on motion of the shoulder when the pain became severe. The *x*-ray plate (Fig. 109) showed a mass about the acromioclavicular joint with possibly slight atrophy of the acromion and the acromial end of the clavicle. A tenative diagnosis of tuberculous abscess was made.

Operation was performed on July 29, 1914, by Dr. Gill. A curved incision was made in front of the tumor and a skin flap

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[1] Read before the Philadelphia Academy of Surgery, May 3, 1915. Reprinted from Annals of Surgery, 1915, lxii, 634.

dissected upward. The tumor lay close beneath the skin and was soon penetrated. A mass of soft tissue resembling a soft blood clot in consistency but grayish in color oozed out abundantly. Hemorrhage was quite free. The tumor mass was cleaned out as thoroughly as possible with finger, sponges, and curette. It seemed to be enclosed in front, at least, by a thin capsule lined with large veins. The lower surface of the acromial end of the clavicle felt roughened, otherwise there appeared to be no bony origin of the tumor. Bleeding was controlled by packing tightly with iodoform gauze and a few sutures were placed to close the incision in part. Diagnosis was made of sarcoma of the clavicle. Microscopic section of the tumor showed it to be of the spindle-celled variety.

The use of Coley's fluid was begun shortly after the operation and was continued in ascending doses until considerable reaction was produced. The wound healed on August 18, 1914. But in less than 2 months the tumor gave evidence of recurrence (Figs. 110 and 111). On November 7, 1914, Dr. Ashhurst operated and removed in one mass a third to a half of the clavicle, the acromion, and the coracoid process together with the surrounding soft tissues. More of the deltoid and trapezius muscles were then removed until no evidence of tumor remained. The skin was approximated and sutured with the arm abducted to a right angle.

The use of Coley's fluid was continued, and the wound was healed by December 19, 1914 (Fig. 112). About the middle of January the tumor appeared to be recurring in the trapezius and the scapula.

On February 8, 1915, the tumor appeared as shown in Fig. 113. At this time the mass was dark in color, owing to hemorrhage within the tumor substance. She was readmitted for operation. Spontaneous rupture of the tumor occurred after her admission and about a pint of blood was lost before the hemorrhage could be controlled by packing.

On February 17, 1915, Dr. Ashhurst performed the third operation consisting of complete excision of the scapula with the exception of the lower angle and the glenoid, and of the trapezius half way to the occiput. Very little hemorrhage occurred owing to the fact that the line of incision was carried wide of the tumor. Part of the clavicle was removed although it did not appear to be involved in the tumor. The brachial plexus was freely exposed, but was not injured in the operation. The skin was approximated and sutured again with the arm in abduction.

The patient failed to recover consciousness after the operation

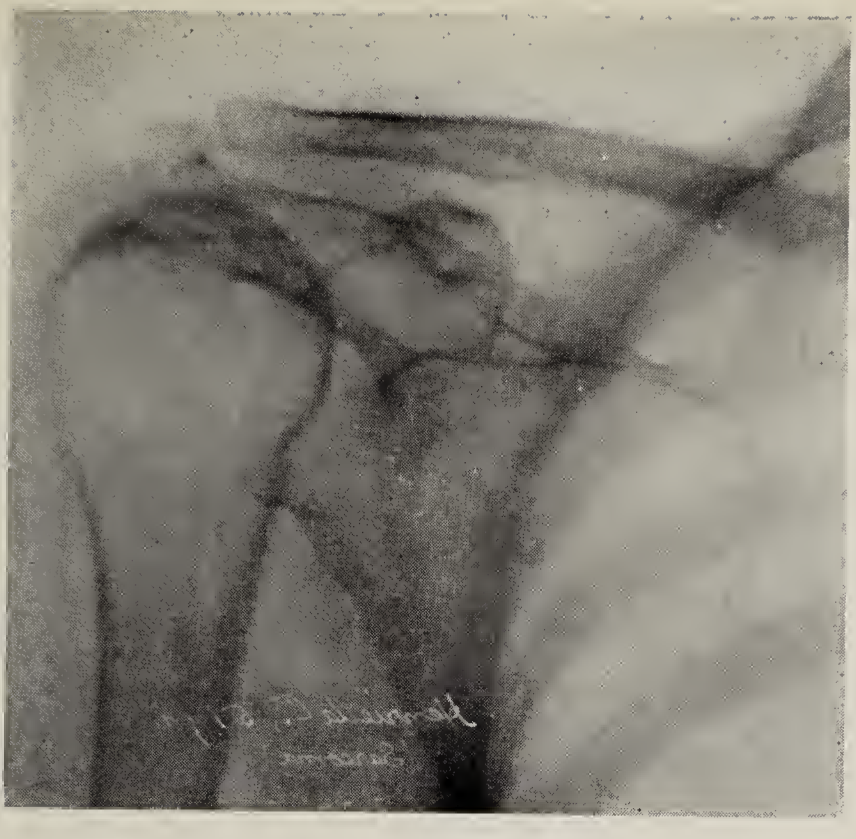

FIG. 109.—Sarcoma of clavicle, July, 1914.

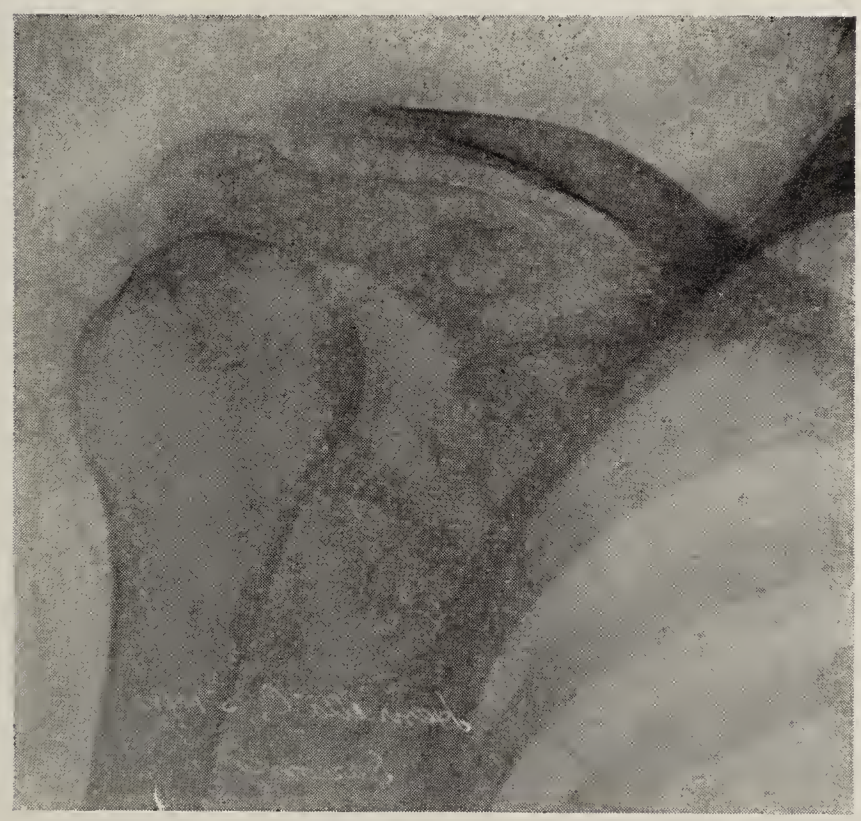

FIG. 110.—Sarcoma of clavicle (November, 1914) three and one-half months after first operation.

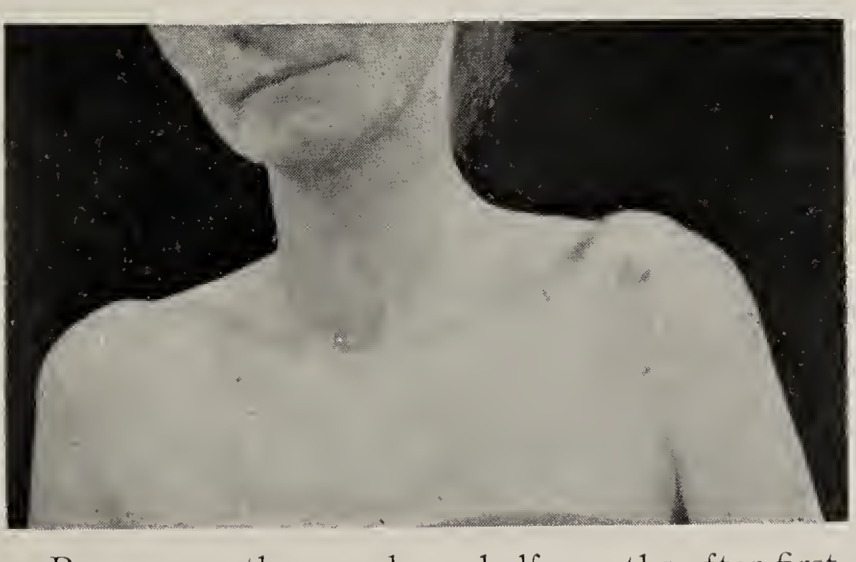

FIG. 111.—Recurrence three and one-half months after first operation (November 5, 1914).

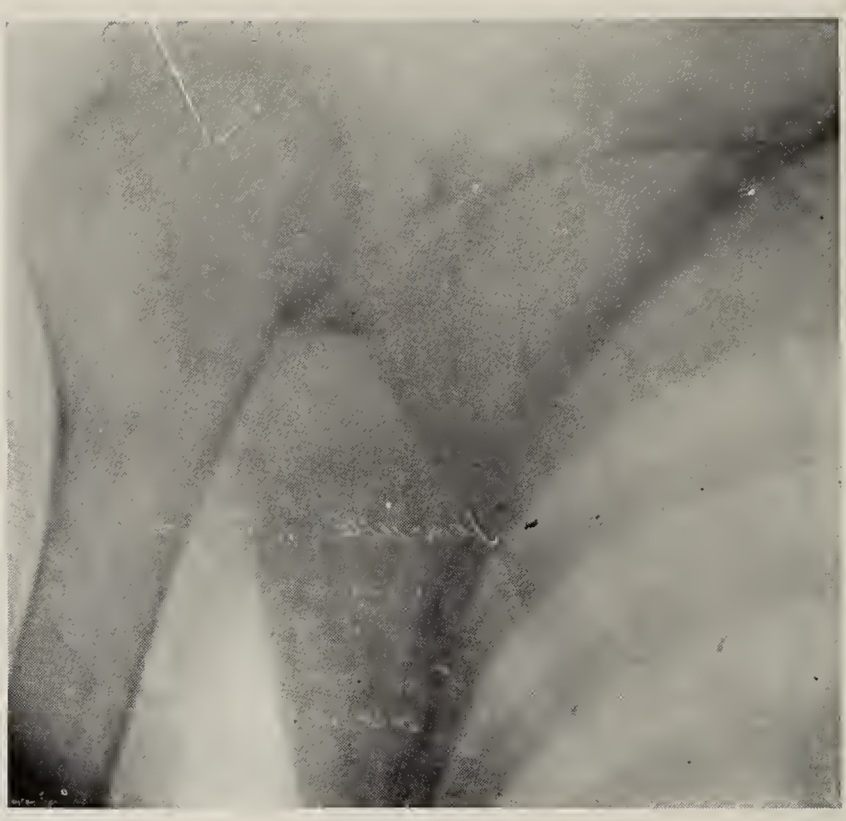

FIG. 112.—Sarcoma of clavicle (December 19, 1914) after excision of outer end of clavicle, acromion and coracoid.

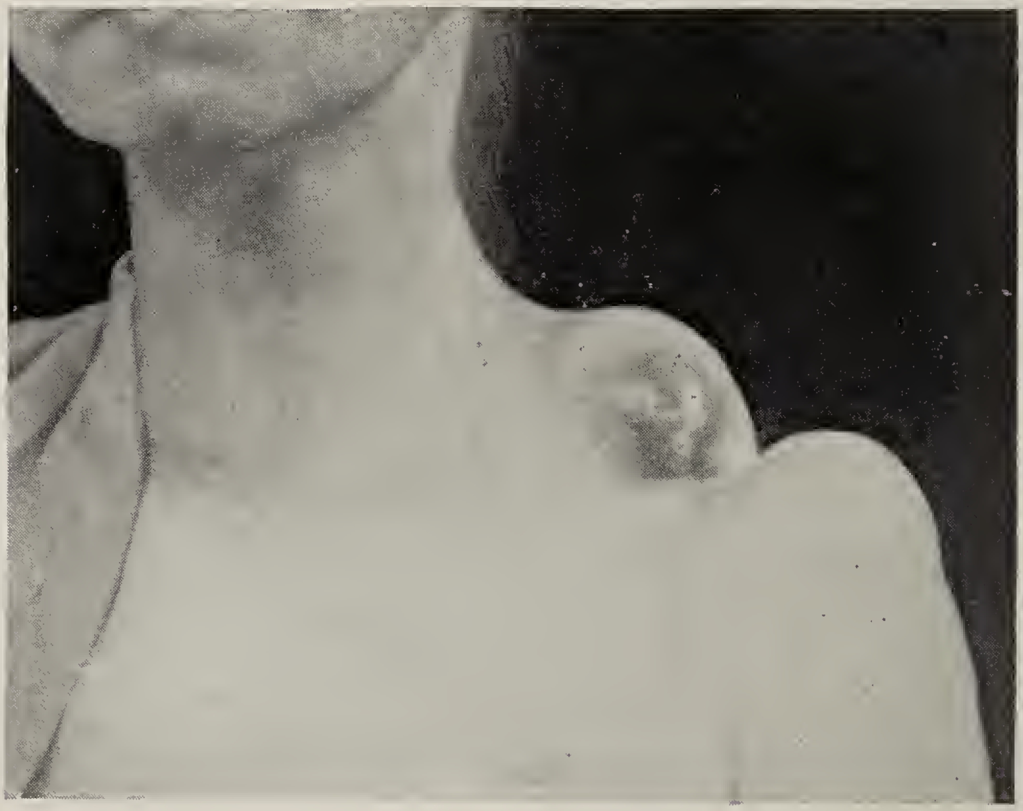

FIG. 113.—Recurrence three months after second operation (February 8, 1915).

and died three hours later. Autopsy showed sarcomatous metastases in the lungs.

Coley has reported 10 cases of sarcoma of the clavicle. Of these 8 gave a distinct history of antecedent trauma. In the case recorded above there is no such history. Coley in his report (*Annals of Surg.*, 1913, lviii, p. 556) of a case discusses the literature briefly and confirms his conclusions expressed in 1910 that:

1. Primary sarcoma of the clavicle is one of the most malignant of all neoplasms.
2. While a rare condition, it requires early diagnosis and very radical treatment.
3. The mortality of the operation itself should be small.
4. The danger of local and general metastases is very great.
5. The use of Coley's fluid is strongly indicated as a routine measure immediately after operation.

# FRACTURE OF THE BASE OF THE SKULL WITH ESCAPE OF CEREBROSPINAL FLUID FROM THE EAR. THE EFFECT OF ATROPIN AND EPINEPHRIN UPON THE SECRETION[1]

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The purpose of this paper is threefold:

1. To report a case of fracture of the base of the skull with the escape of an unusual amount of cerebrospinal fluid from the ear.

2. To outline briefly the evolution of the hypothesis that the cerebrospinal fluid is a true secretion.

3. To present the effect both chemically and clinically of the hypodermic injection of atropin and epinephrin upon the secretion of cerebrospinal fluid.

Fracture of the base of the skull with the escape of cerebrospinal fluid from the cranium has been observed for centuries. Bidloo the elder (quoted by St. Clair Thomson), writing in the latter half of the seventeenth century, reported a case of a patient who had a fracture of the bridge of the nose followed by a continuous flow of clear watery fluid from the right nostril; twenty ounces of this fluid escaped in twenty-four

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[1] I have to thank Dr. Charles H. Frazier most cordially in making it possible for me to carry out the work presented in this paper, and freely placing at my disposal valuable unpublished data concerning the effects of the injection of certain saline extracts of the body organs upon the secretion of cerebrospinal fluid in dogs.

Extracted from the American Journal of the Medical Sciences, April, 1915, No. 4, vol. cxlix, p. 580.

hours. Stalpartius von der Wiel, in 1725, published a case in which a large quantity of clear watery fluid escaped from the ear after a severe injury to the head. Matthein and others have collected a number of such cases. Indeed, judging from the recent literature, surgeons do not look upon this as a matter of rare occurrence.

The case which we have to report is not a unique one by any means; however, judging from his age, weight, and stature, the amount of cerebrospinal fluid that escaped from the ear at regular intervals is rather remarkable.

Case I.—W. R., Episcopal Hospital, aged six years, male; weight thirty-five pounds.

Eighteen hours before admission into the hospital the child fell down a flight of stairs backward while attempting to catch a ball that he had thrown upward. The screams attracted the mother's attention. She found the child lying on the floor, in an unconscious condition, bleeding from the left ear. Within a few minutes blood and clear colored fluid flowed alternately from the left ear. Later (about twenty minutes) the blood ceased entirely, but the clear colored fluid continued to flow, with occasional momentary stoppage up to the time of admission into the hospital. The child remained unconscious for six hours after the accident; thereafter was conscious of things around him. He vomited just after the fall and again six hours later.

The child was of instrumental delivery. Since the age of eight months the patient has had frequent epileptic seizures, the attacks occurring at irregular intervals. The child may go as long as three days without a seizure, on the other hand the attacks may occur daily, or several times during the day, and continue in this manner for two or three days. The oncoming attack is usually ushered in by the child being peevish, fretful, restless, spitting, etc. Then comes the seizure which manifests itself by a rapid contraction of arms and legs, a sudden scream and a falling to the floor, rapidly becoming cyanotic and blue in the face. At times the child froths at the mouth but never bites his tongue. The attack is usually three minutes in duration. The child, according to the parents, is bright and has a wonderful memory. No history of mental deterioration. Had a discharging right ear two weeks ago which rapidly cleared up under treatment. Mother states that the child did not have an epileptic seizure before the fall, nor were there any symptoms of an attack following the accident.

On admission to the hospital, eighteeen hours after the accident, the examination revealed a fairly well-nourished child of six years, somewhat restless, but nothing more than one would expect of a child in a strange place. The child would heed immediately when his name was called, and would protrude the tongue or raise the arm when asked to; we were unable to draw the patient into a conversation, but he would at intervals articulate words, as water, mother, and the like. However, he would never utter a complete sentence.

Aside from the discharging left ear the physical examination was essentially negative. There were no signs of injury to the scalp. Eyes reacted promptly to light, pupils were equal. Ocular movements normal. Cardiac action was rapid, pulse of good tension and volume. Lungs, abdomen, and extremities were negative.

The patella, achilles, biceps, and triceps reflexes reacted promptly but were not increased. There were no palsies, no ankle-clonus. Skin reflexes and sensation normal.

Temperature, 97° F.; pulse, 130; respiration, 24.

From the left ear could be seen a continuous flow of clear watery fluid. The flow was so rapid that within a short time the pillow-cover in the region of the head would be soaked with the fluid. The fluid reduced Fehling's solution. Specific gravity, 1007. One c.mm. showed 1730 red blood corpuscles, 49 polymorphonuclear leukocytes, and 39 lymphocytes.

March 16. The child is quiet, lies with the right side of the head on the pillow. The left ear continues to discharge a clear watery fluid at irregular intervals; that is to say, the flow ceases an hour or more, two or three times in the twenty-four hours.

The child had a convulsion which lasted two minutes; during the convulsion the lips became cyanosed, face pinched, upper and lower extremities contracted rapidly. There was no frothing at the mouth nor biting of the tongue. Had to be catheterized. Temperature, 103° F.; pulse, 135; respiration, 24.

March 17. Very little change in condition. The child drinks a great deal of water and takes liquid diet well.

The ear continues to discharge at irregular intervals. Had a convulsion, similar to the one on the previous day. No record was made of the amount of secretion during the convulsion, but the attending nurse states that there was not a noticeable increase during the convulsion, judging from the wet spot on the pillow cover.

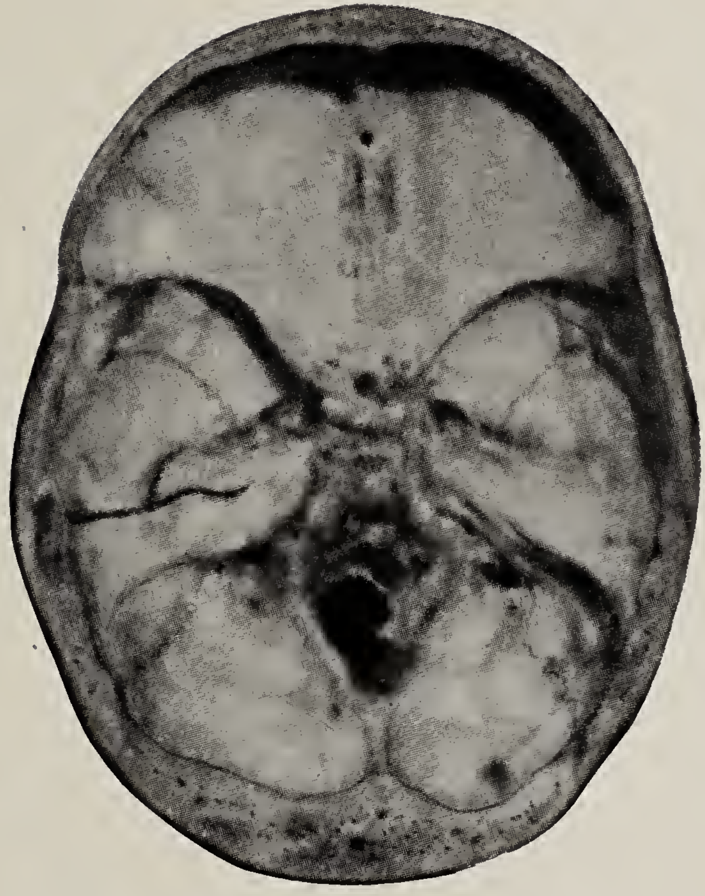

FIG. 114.—Showing the Y-shaped fracture involving the petrous portion of the left temporal bone. The fracture was outlined with lead-pencil and a photograph taken of the base of the skull.

March 18. The ear ceased secreting only once during the twenty-four hours, and that was for about three hours, while the child was asleep. There is a suggestive Kernig and reflex resistance of the neck muscles to flexion of head. The right ear began to discharge mucopurulent material. Secretion collected from the left ear by means of an eye dropper in sixty minutes was 15 c.c.

The eye-grounds were examined by Dr. Bromley. The report was as follows:

O. D. Nerve circumscribed and flat; very little venous congestion. Media clear.

O. S. Slight engorgement of veins, nerve perfectly flat and circumscribed. No edema of retina. Media clear.

March 19. Patient takes water and liquid nourishment freely. The fluid collected from the ear in sixty minutes amounted to 21 c.c. The child dropped off to sleep and the ear stopped secreting for hours. On awakening the ear began secreting again.

Ten grains of urotropin were given, and fluid collected every five minutes for two hours and placed in separate test-tubes. We were unable to demonstrate the presence of urotropin with the bromine water test in the fluid collected.

Blood Count. White blood corpuscles, 17,200; polynuclears, 66⅖ per cent., mononuclears, 3 per cent.; transitionals, 7⅖ per cent.; lymphocytes, 22⅕ per cent.; eosinophiles, 1 per cent.

March 21. Very little change in condition of patient. Kernig's sign suggestive. No rigidity of neck.

March 23. Child is quiet most of the time, occasionally becoming restless. Kernig is positive. No rigidity of neck; no bowing of back.

March 24. The child is quite restless at times. There is rigidity of the neck muscles, with slight retraction of the head. Kernig's sign is positive. No bowing of the back. Pulse is rapid; secretion from left ear is scanty.

Blood Count. White blood corpuscles, 25,350; polynuclears, 79 per cent.; transitionals, 3 per cent.; lymphocytes, 18 per cent.

March 25. The patient appears to be in great agony, crying out at frequent intervals; retraction of neck is marked; there is also bowing of the back, with drawing up of lower extremities. The left ear has ceased secreting entirely. Skin is dry; eyes sunken; tongue parched; radial pulse scarcely perceptible.

From this point the patient rapidly went downward, and died March 28.

The autopsy was perfromed by Dr. C. Y. White, director of the pathological laboratory of the Episcopal Hospital. He reports as follows:

The dura is transparent, the inner surface being smooth and glistening. The vessels of the pia mater are only slightly congested. No macroscopic evidence of a meningitis. No edema beneath the pia.

On severing the brain from the cord an excessive amount of cerebrospinal fluid escaped into the base of the skull.

The dura is entirely stripped for the purpose of examining the skull, and there is found a fracture involving the squamous and anterior surface of the petrous portion of the left temporal bone. That part of the fracture involving the petrous portion is Y-shape, and extends into the internal auditory meatus. One limb of the Y begins with the depression for the Gasserian ganglion; the other with the opening for the small petrosal nerve. The two uniting just external to the Hiatus Fallopii and running outward parallel with and $\frac{1}{8}$ of an inch beneath the superior border to the squamous portion, where it continues directly upward in that bone for one inch.

Dr. W. B. Cadwalader made numerous sections of the brain and choroid plexus, and submits the following report:

Sections were made from various parts of the cerebral cortex and the cerebellum. For the most part they were entirely negative, but here and there in some sections a few polymorphonuclear leukocytes could be seen infiltrating the pia, and grouped about some of the smaller bloodvessels within the fissures of the cortex. This was a real meningitis, yet it was so slight that it would require more than a superficial examination to find it. Sections from the basal ganglia, internal capsule, pons, cerebellum, and different levels of the spinal cord appear to be entirely normal. There were no evidences of meningitis in these parts and the tissue stained normally with the Weigert stain. Sections of the choroid plexus were made and were stained by a number of different methods. All the cells appeared entirely normal. I could not demonstrate some of the granules which are thought to react differently to stains, and which have been supposed to have some relationship to secretory functions. Comparing the sections taken from this case with similar sections of two other brains which ought not to have had any disease of the choroid plexus, I could not really find any difference. On the whole it is just to conclude that this specimen, brain and cord, shows absolutely nothing abnormal except a very mild and diffuse infiltration of the pia which was probably of recent origin.

THE EVOLUTION OF THE HYPOTHESIS THAT THE CEREBROSPINAL FLUID IS A TRUE SECRETION. The hypothesis that the cerebrospinal fluid is a secretory product of the choroid plexus has been the source of infinite research for a number of decades. Leonard Hill,[1] in the early 90's, advocated the hypothesis that the fluid is a serous transudation from the capillaries of the vascular choroid. He regards the cerebrospinal fluid as the lymph of the brain, its secretion and resorption being explained on the same lines as other lymphatic exudate. He contends that the secretion of the fluid increases with the rise in cerebral capillary pressure and the latter varies directly and absolutely with vena cava pressure. He cites a case of St. Clair Thomson's in which there was a continuous flow of cerebrospinal fluid from the left nostril. At the invitation of St. Clair Thomson[2] he made the following observations:

I

1. Patient sitting quietly, and in five minutes 23 m. were collected.
2. Patient sitting and straining, and in five minutes 35 m. were collected.
3. Patient sitting quietly, and in five minutes 19 m. were collected.
4. Patient sitting and straining, and in five minutes 33 m. were collected.
5. Patient lying—lying down and not straining, 27 m. were collected.
6. Patient sitting with head overhanging, in five minutes 28 m. were collected.
7. Patient sitting with head forward, and in five minutes 15 m. were collected.

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[1] The Cerebrospinal Fluid, Allbutt's System of Medicine, 1899, vii, 239.

[2] Ibid.

## II

1. Abdomen compressed, and in five minutes 27 m. were collected.
2. Sitting quietly, and in five minutes 14 m. were collected.
3. Abdomen compressed, and in five minutes 24 m. were collected.
4. Sitting quietly, and in five minutes 23 m. were collected.

The experiments carried out by Hill are valueless, owing to the short space of time given to each procedure.

It is stated that the normal amount of cerebrospinal fluid present in the subarachnoid space is about 100 c.c. That this fluid is rapidly formed is shown by the enormous amount of fluid escaping from the ear of my patient. It is clear that any condition which would cause congestion of the cerebral vessels would mechanically encroach upon the subarachnoid space, thereby increasing intracranial pressure. A sudden increase of flow after straining or compressing the abdomen, and a decrease in flow when the mechanical pressure is released is to be expected. Suffice it to say, if the straining or compression of the abdomen had been kept up for twenty to thirty minutes, Hill's observations would have been different, as will be shown later. Furthermore, Sabin has shown that the subarachnoid space is fully developed, long before the lymphatics have budded out from the subclavian and iliac veins.

Mott[1] is credited with giving the following compositions of the fluid, which he considered to be against its being a transudation from the blood or a lymphatic secretion.

1. It contains 0.02 per cent. of proteins against 7 per cent. in blood plasma and 4.5 per cent. in body lymph.
2. There is an absence of lipochroma.
3. There are no leukocytes in normal cerebrospinal fluid.
4. In enteric fever there is an absence of agglutination.

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[1] The Physical and Chemical Properties of the Cerebrospinal Fluid. Oliver Sharpey's Lectures, Lancet, July 2 and 9, 1910.

5. It has no hemolytic action on the blood corpuscles of other animals.

6. It contains no alexins.

Willis, in 1664, first mentioned the glandular appearance of what is now called the choroid plexus. Faivre, in 1853, maintained that the cuboidal cells which cover the choroid plexuses are secreting cells concerned in the formation of cerebrospinal fluid. Mott examined histologically the choroid plexus of a human subject. He reports that the sections under high power reveal tufts of vessels surrounded by a loose connective tissue covered by a single layer of cubical, spheroidal or polyhedral cells lying on a basement membrane. Around the arteries and arterioles and lying in the loose connective tissue numerous nerve fibers are seen in the form of a plexus. In the sections he also observed vacuoles in the cells, and cup-like cavities in others, as one would expect if fluid had escaped from the vacuole. He concludes, therefore, from the histological evidence that the choroid plexus is a gland with an external secretion, but with an internal destination. Being a mixed gland, he suggests that its mode of formation is in an inverse manner to those formed by epithelial invagination of glands with an excretory duct. Ependymal invagination occurs for the choroid plexus.

Kramer,[1] describes the histological appearance of the choroid plexus as highly vascular projections of the pia mater into the ventricle, covered with villous-like projections about 1 to 2 mm. in diameter; under the microscope these villi are seen to be made up of a number of secondary villi about 25 mm. in diameter, which again show grape-like projections. Through the centre of the villi run comparatively thick-walled bloodvessels, giving the organ a highly vascular appearance. The connective tissue of the villi is that of the pia mater. Covering the villi of the plexus is a layer of spheroidal cells in each of which may be seen in addition to the nucleus, yellowish granules. These cells are probably the secreting cells of the organ. These cells are without ducts, pouring their secretions directly into the ventricular system of the brain.

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[1] Fall of Pressure Produced by Choroid Extract, Jour. Am. Med. Assn., 1911, lvi, 265.

Carl Schmidt, in 1850, advanced the chemical evidence in favor of a true secretion. Petit and Girard[1] in 1902, demonstrated the secretory functions of the choroid plexus in different classes of vertebrates. They found that administration of substances endowed with hypersecreting action caused histological changes in the cells covering the plexus. Dixon and Halliburton have experimental proof that an intravenous injection of a extract of choroid plexus produces an increased secretion of the cerebrospinal fluid. The injections of extracts of the brain produces the same effect upon the respiration, blood-pressure and flow of the cerebrospinal fluid, as does the injection of extract of choroid plexus. So also in cases of general paralysis and brain softening, where destructive processes are actively taking place, the cerebrospinal fluid when injected, produces identical effects. They suggest, therefore, that the increase in output of fluid is due to the stimulating action of some chemical substance (hormone) in the extract on the secreting cells of the gland, and that the hormone is a product of the brain's metabolism which passes to the choroid plexus and stimulates the secreting cells of the plexus.[2]

Writers as Levy, Cushing, Obersteiner, Cavazzani, Frazier, and others likewise maintain the secretory activity of the choroid plexus as the formation of cerebrospinal fluid.

The observations in Table I were made in the case of the patient herein reported.

The table summarizes the amount of fluid excreted in five consecutive minutes, the state of the patient, whether quiet, restless or apparently asleep and the heart rate. (The heart

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[1] Smith, E., The Cerebrospinal Fluid, Guy's Hosp. Gaz., London, 1911, xxv, 141.

[2] W. Mestrezat, in a recent publication, expresses his belief that the cerebrospinal fluid is neither a transudation nor a secretion, but is a product of dialysis from the blood plasma.

TABLE I

| Time | Amount collected in five minutes approximate in c.c. | State of patient. | Heart rate. | Analysis of cerebrospinal fluid. | | | | | Expressed as grams per 100 c.c. fluid. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Duration, minutes. | Actual am't, c.c. | Solids, gm. | Ash gm. | $K_2O$ gm. | Solids. | Ash. | $K_2O$. |
| | Period I | Control | | | | | | | | | |
| 11.05 | .73 c.c. (12 m.) | Apparently asleep | 126 | 30 | 5.4 | 0.0720 | 0.0494 | 0.0008 | 1.35 | 0.91 | 0.015 |
| 11.10 | 1.54 c.c. (25 m.) | Apparently asleep | 130 | | | | | | | | |
| 11.15 | 1.23 c.c. (20 m.) | Apparently asleep | 128 | | | | | | | | |
| 11.20 | .48 c.c. ( 8 m.) | Apparently asleep | 130 | | | | | | | | |
| 11.25 | .49 c.c. ( 8 m.) | Apparently asleep | 128 | | | | | | | | |
| 11.30 | .86 c.c. (14 m.) | Apparently asleep | 126 | | | | | | | | |
| A.M. | Period II[1] | | | | | | | | | | |
| 5 | .73 c.c. (12 m.) | Restless and quiet | 170 | 40 | 4.4 | 0.0676 | 0.0476 | 0.0009 | 1.54 | 1.08 | 0.017 |
| 10 | .37 c.c. ( 6 m.) | Restless and quiet | 140 | | | | | | | | |
| 15 | .30 c.c. ( 5 m.) | Restless and quiet | 163 | | | | | | | | |
| 20 | .37 c.c. ( 6 m.) | Restless and quiet | 134 | | | | | | | | |
| 25 | .73 c.c. (12 m.) | Restless | 170 | | | | | | | | |
| 30 | .55 c.c. ( 9 m.) | Restless and quiet | 147 | | | | | | | | |
| 35 | .37 c.c. ( 6 m.) | Quiet | 136 | | | | | | | | |
| 40 | .86 c.c. (14 m.) | Quiet | 140 | | | | | | | | |
| | Period III | | | | | | | | | | |
| 45 | .96 c.c. (16 m.) | Quiet | 140 | 25 | 7.2 | 0.0751 | 0.0501 | 0.0009 | 1.04 | 0.70 | 0.013 |
| 50 | 1.50 c.c. (25 m.) | Restless and quiet | 136 | | | | | | | | |
| 55 | 1.38 c.c. (23 m.) | Quiet | 136 | | | | | | | | |
| 60 | 1.68 c.c. (28 m.) | Restless and quiet | 168 | | | | | | | | |
| 65 | 1.38 c.c. (23 m.) | Restless | 168 | | | | | | | | |
| | Period IV | | | | | | | | | | |
| 70 | 1.20 c.c. (20 m.) | Restless | 164 | 20 | 4.75 | 0.0587 | 0.0379 | 0.0006 | 1.24 | 0.80 | 0.013 |
| 75 | 1.14 c.c. (19 m.) | Restless | 164 | | | | | | | | |
| 80 | 1.02 c.c. (17 m.) | Restless | 164 | | | | | | | | |
| 85 | 1.20 c.c. (20 m.) | Restless | 166 | | | | | | | | |
| | Period V | | | | | | | | | | |
| P.M. | Not measured | | ... | 90 | Not measured | 0.0614 | 0.0392 | 0.0007 | | | |
| 2.35 | Period VI | | | | | | | | | | |
| 5 | 1.14 c.c. (19 m.) | Quiet | 140 | 30 | 7.0 | 0.0648 | 0.0414 | 0.0006 | 0.92 | 0.59 | 0.009 |
| 10 | 1.14 c.c. (19 m.) | Quiet | 138 | | | | | | | | |
| 15 | 1.20 c.c. (20 m.) | Quiet and restless | 145 | | | | | | | | |
| 20 | .96 c.c. (16 m.) | Quiet | 138 | | | | | | | | |
| 25 | 1.02 c.c. (17 m.) | Quiet | 140 | | | | | | | | |
| 30 | 1.20 c.c. (20 m.) | Quiet | 140 | | | | | | | | |
| | Period VII | | | | | | | | | | |
| 35 | 1.08 c.c. (18 m.) | Apparently asleep | 140 | 30 | 7.1 | 0.0930 | 0.0624 | 0.0011 | 1.31 | 0.88 | 0.016 |
| 40 | .96 c.c. (16 m.) | Apparently asleep | 138 | | | | | | | | |
| 45 | 1.14 c.c. (19 m.) | Quiet | 140 | | | | | | | | |
| 50 | 1.14 c.c. (19 m.) | Quiet | 138 | | | | | | | | |
| 55 | 1.38 c.c. (23 m.) | Restless | 144 | | | | | | | | |
| 60 | 1.14 c.c. (19 m.) | Restless | 144 | | | | | | | | |
| | Period VIII | | | | | | | | | | |
| | Not measured | | ... | 65 | 13 | 0.1530 | 0.1022 | 0.0014 | 2.73 | 1.82 | 0.025 |
| P.M. | Period IX | | | | | | | | | | |
| 5 | .30 c.c. ( 5 m.) | Restless | 150 | 110 | 6.9 | 0.1096 | 0.0632 | 0.010 | 1.59 | 0.92 | 0.014 |
| 10 | .12 c.c. ( 2 m.) | Apparently asleep | 140 | | | | | | | | |
| 15 | 0.0 | Apparently asleep | 138 | | | | | | | | |
| 20 | 0.0 | Apparently asleep | 136 | | | | | | | | |
| 25 | 0.0 | Apparently asleep | 136 | | | | | | | | |
| 30 | 1.02 c.c. (17 m.) | Restless | 140 | | | | | | | | |
| 110 | 5.76 c.c. (96 m.) | | | | | | | | | | |
| | Period X | | | | | | | | | | |
| 5 | .96 c.c. (16 m.) | Apparently asleep | 150 | 45 | 13.4 | 0.1612 | 0.1065 | 0.0015 | 1.20 | 0.80 | 0.011 |
| 10 | 1.20 c.c. (20 m.) | Apparently asleep | 140 | | | | | | | | |
| 15 | 1.14 c.c. (19 m.) | Apparently asleep | 136 | | | | | | | | |
| 20 | 1.32 c.c. (22 m.) | Apparently asleep | 138 | | | | | | | | |
| 25 | 2.40 c.c. (40 m.) | Apparently asleep | 138 | | | | | | | | |
| 30 | 1.38 c.c. (23 m.) | Restless | 140 | | | | | | | | |
| 35 | 1.80 c.c. (30 m.) | Apparently asleep | 140 | | | | | | | | |
| 40 | 1.38 c.c. (23 m.) | Apparently asleep | 140 | | | | | | | | |
| 45 | 1.32 c.c. (22 m.) | Restless | 150 | | | | | | | | |

[1] Epinephrin (1 to 1000) 5 m. injected subcutaneously. A blanching appeared at point of injection.

TABLE I—Continued

| Time | Amount collected in five minutes approximate in c.c. | State of patient. | Heart rate. | Analysis of cerebrospinal fluid. | | | | | Expressed as grams per 100 c.c. fluid. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Duration, minutes. | Actual am't, c.c. | Solids, gm. | Ash gm. | $K_2O$ gm. | Solids. | Ash. | $K_2O$. |
| | Period XI | | | | | | | | | | |
| 5 | .80 c.c. (15 m.) | Quiet | 144 | | | | | | | | |
| 10 | 1.20 c.c. (20 m.) | Restless | 150 | | | | | | | | |
| 15 | 1.14 c.c. (19 m.) | Quiet | 142 | | | | | | | | |
| 20 | 1.14 c.c. (19 m.) | Quiet | 140 | | | | | | | | |
| 25 | 1.02 c.c. (17 m.) | Quiet | 138 | | | | | | | | |
| 30 | .96 c.c. (16 m.) | Quiet | 150 | | | | | | | | |
| 35 | 1.14 c.c. (19 m.) | Restless | 154 | | | | | | | | |
| 40 | .90 c.c. (15 m.) | Quiet | 150 | | | | | | | | |
| | | | | 75 | 14.5 | 0.1607 | 0.0934 | 0.0013 | 1.10 | 0.62 | 0.009 |
| 45 | .84 c.c. (14 m.) | Quiet | 157 | | | | | | | | |
| 50 | .96 c.c. (16 m.) | Restless | 170 | | | | | | | | |
| 55 | .84 c.c. (14 m.) | Restless | 168 | | | | | | | | |
| 60 | .60 c.c. (10 m.) | Restless | 168 | | | | | | | | |
| 65 | .60 c.c. (10 m.) | Restless | 166 | | | | | | | | |
| | | Was given water | | | | | | | | | |
| 70 | .84 c.c. (14 m.) | Quiet | 150 | | | | | | | | |
| 75 | .84 c.c. (14 m.) | Apparently asleep | 140 | | | | | | | | |
| | Period XII | | | | | | | | | | |
| 80 | 1.80 c.c. (30 m.) | Apparently asleep | 140 | | | | | | | | |
| 85 | 2.10 c.c. (35 m.) | Apparently asleep | 138 | | | | | | | | |
| 90 | .84 c.c. (14 m.) | Apparently asleep | 140 | | | | | | | | |
| 95 | 1.20 c.c. (20 m.) | Restless | 140 | | | | | | | | |
| 100 | .90 c.c. (15 m.) | Restless | 144 | 45 | 12.4 | 0.1131 | 0.645 | 0.0008 | 0.91 | 0.52 | 0.006 |
| 105 | .84 c.c. (14 m.) | Quiet | 150 | | | | | | | | |
| 110 | 1.14 c.c. (19 m.) | Restless | 154 | | | | | | | | |
| 115 | 1.50 c.c. (25 m.) | Quiet and restless | 145 | | | | | | | | |
| 120 | 1.50 c.c. (25 m.) | Quiet and restless | 148 | | | | | | | | |

AVERAGE OF PERIOD

| | Solids. | Ash. | $K_2O$ |
|---|---|---|---|
| I | 1.35 | 0.91 | 0.015 |
| II to X (except V) | 1.38 | 0.91 | 0.014 |
| XI and XII | 1.02 | 0.59 | 0.008 |

PERCENTAGE OF ASH IN TERMS OF TOTAL SOLIDS

| | | | | | |
|---|---|---|---|---|---|
| I | 68.6 | V | 63.9 | IX | 57.8 |
| II | 71.9 | VI | 63.9 | X | 66.1 |
| III | 66.7 | VII | 67.3 | XI | 58.1 |
| IV | 64.6 | VIII | 66.8 | XII | 57.0 |

AVERAGE

| | | | | | |
|---|---|---|---|---|---|
| I | 68.6 | II to X (inc. V) | 65.3 | XI to XII | 57.7 |

rate was taken twice in each five-minute period, with a stethoscope, and the average recorded.) The fluid was collected in sterile glass test-tubes, labeled periods I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII. The tubes were given to Dr. Charles H. Frazier, who in turn secured the valuable service of Dr. A. E. Taylor, professor of physiological chemistry in the University of Pennsylvania, for chemical analysis.

## TABLE II

At 3.35 water, ʒiv, given by mouth, and twenty-five minutes later atropin sulphate, gr. $\frac{1}{200}$, hypodermically.

| Time. | Approximate amount in c.c. | State of patient. | Heart rate. |
|---|---|---|---|
| 2:35 | | | |
| 5 | 2.70 c.c. (45 m.) | Quiet | 134 |
| 10 | 2.70 c.c. (45 m.) | Quiet | 130 |
| 15 | 2.40 c.c. (40 m.) | Quiet | 134 |
| 20 | 2.70 c.c. (45 m.) | Quiet | 132 |
| 3:00 | Atropin, 0.05 gr. hypodermically. | | |
| 5 | 2.40 c.c. (40 m.) | Restless | 140 |
| 10 | 1.50 c.c. (25 m.) | Restless | 145 |
| 15 | 1.50 c.c. (25 m.) | Quiet | 138 |
| 20 | 1.50 c.c. (25 m.) | Quiet | 136 |
| 25 | .96 c.c. (16 m.) | Quiet and restless | 138 |
| 30 | 1.14 c.c. (19 m.) | Quiet and restless | 136 |
| 35 | 1.32 c.c. (22 m.) | Quiet and restless | 138 |
| 40 | 1.08 c.c. (18 m.) | Apparently asleep | 138 |
| 45 | 1.26 c.c. (21 m.) | Apparently asleep | 140 |
| 50 | 1.14 c.c. (19 m.) | Apparently asleep | 138 |
| 55 | .99 c.c. (15 m.) | Apparently alseep | 144 |
| 60 | .90 c.c. (15 m.) | Apparently asleep | 138 |
| 65 | .96 c.c. (16 m.) | Apparently asleep | 144 |
| 70 | .60 c.c. (10 m.) | Apparently asleep | 140 |
| 75 | .90 c.c. (15 m.) | Apparently asleep and restless | 144 |
| 80 | 1.02 c.c. (17 m.) | Apparently asleep | 138 |
| 85 | .90 c.c. (15 m.) | Apparently asleep | 138 |
| Milk is given. | | | |
| 90 | .66 c.c. (11 m.) | Restless | 145 |
| 95 | 1.38 c.c. (23 m.) | Restless | 146 |
| 100 | 1.32 c.c. (22 m.) | Restless | 144 |
| 105 | —— | Exceedingly restless | . . . |
| 110 | .96 c.c. (16 m.) | Restless | 146 |
| 115 | .96 c.c. (16 m.) | Restless | 144 |
| 120 | .90 c.c. (15 m.) | Restless | 150 |

## TABLE III.—TO SHOW THE INFLUENCE OF SLEEP, QUIET, AND RESTLESSNESS UPON THE SECRETION OF CEREBROSPINAL FLUID, MARCH 22

| Time. | Approximate amount collected in 5 minutes in c. c. | State of patient. | Heart rate. |
|---|---|---|---|
| 11:00 | .30 c.c. ( 5 m.) | Quiet | 134 |
| 5 | 1.08 c.c. (18 m.) | Quiet and restless | 134 |
| 10 | 1.08 c.c. (18 m.) | Quiet | 135 |
| 15 | .60 c.c. (10 m.) | Restless and quiet | 145 |
| 20 | .90 c.c. (15 m.) | Quiet | 134 |
| 25 | .90 c.c. (15 m.) | Quiet | 140 |
| 30 | .96 c.c. (16 m.) | Quiet | 140 |
| 35 | 1.14 c.c. (19 m.) | Quiet | 138 |
| 40 | 1.38 c.c. (23 m.) | Quiet and restless | 144 |
| 45 | 1.14 c.c. (19 m.) | Quiet and restless | 142 |
| 50 | 1.08 c.c. (18 m.) | Quiet | 134 |
| 55 | 1.50 c.c. (25 m.) | Quiet | 136 |
| 60 | 1.14 c.c. (19 m.) | Quiet and restless | 140 |
| 65 | 1.08 c.c. (18 m.) | Quiet and restless | 144 |

### COLLECTED THE FOLLOWING DAY, MARCH 23

| Time. | Approximate amount collected in 5 minutes in c. c. | State of patient. | Heart rate. |
|---|---|---|---|
| 11:00 A.M. | | | |
| 5 | .90 c.c. (15 m.) | Quiet and restless | 140 |
| 10 | .84 c.c. (14 m.) | Quiet and restless | 138 |
| 15 | 1.02 c.c. (17 m.) | Quiet and restless | 140 |
| 20 | 2.28 c.c. (38 m.) | Quiet | 138 |
| 25 | 1.38 c.c. (23 m.) | Quiet and restless | 142 |
| 30 | .90 c.c. (15 m.) | Restless | 150 |
| 35 | .96 c.c. (16 m.) | Restless | 144 |

March 20. Epinephrin (1 to 1000) 5 m. injected subcutaneously into the arm. Eight hours and forty minutes later atropin sulphate, gr. $\frac{1}{250}$, was injected hypodermically. Experiment begun at 11 A.M.

A SUMMARY OF THE EFFECT OF ATROPIN ON CEREBROSPINAL FLOW. Table I. The normal secretion during one hour amounted to 17.7 c.c., average heart rate was equal to 139 beats per minute. After the injection of atropin, gr. $\frac{1}{250}$ hypodermically, the amount of secretion during one hour amounted to 11.8 c.c., a decrease of 5.9 c.c. in one hour. Average heart beat 153 per minute, an increase of 14 beats to the minute.

Table II. The normal secretion for twenty minutes amounted to 10.8 c.c., or at the rate of one hour, 32.3 c.c.; average heart beat per minute 132. After injection of atropin, gr. $\frac{1}{200}$ hypodermically, the amount secreted in the first twenty minutes amounted to 7.1 c.c.; a decrease of 3.7 c.c. in twenty minutes. Average heart rate 138 per minute; an increase of 6 heart beats. The amount secreted in one hour was 16.1 c.c.; a decrease of 16 c.c. for one hour. Average heart beat, 141 per minute; an increase of 9 heart beats per minute. The amount secreted the following thirty minutes at the rate of one hour was 14.1 c.c. The chemical analysis of Table I shows a diminished solid, ash and potassium[1] content with an increased volume of fluid per time unit after injection of atropin. From the above observations it would undoubtedly follow that atropin exerts a partial inhibitory influence upon the secretion of cerebrospinal fluid; that is to say, atropin depresses the nervous stimuli of certain secretory cells of the choroid plexus in the same manner as it does other glands of the body dependent upon nervous stimuli for their secretion. Capelleti has shown that on dogs, atropin and hyoscyamin diminished the flow of cerebrospinal fluid, while pilocarpin increased it. Yoshimura states that atropin produces microscopical changes which are a manifestation of the inhibitory

influence brought to bear on the secretory activity of the cells. Dixon and Halliburton[1] have found that after the administration of atropin, subsequent injection of choroid extract produces an increase of cerebrospinal flow.

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[1] The potassium was determined by the volumetric method of Drushel (cobalt-trinitrite method), and was not done in duplicate, as the amount of fluid was too small.

The decrease in secretion of cerebrospinal fluid can be explained by one of two ways: (1) By granting that there is only one type of secretory nerves in the choroid plexus, and that the activity of the nerve endings depends entirely upon the presence of a chemical substance or hormone, and that the hormone is produced by the choroid cells themselves, atropin[2] would decrease the flow then, by depressing the cells which give rise to the formation of the hormone. The increase in flow following the injection of choroid extract, after the animal has been atropinized would be due to the mechanical replacing of a substance necessary for the stimulation of the cells to secretion. It is not likely that this gland produces its own hormone. We know of no other gland in the body where such a mechanism occurs. It has been shown further that the extract of the brain and cerebrospinal fluid in general paralysis contains a hormone similar to that found in the choroid plexus. (2) It is more plausible to speculate that the gland is supplied with two sets of secretory nerves, namely, those dependent upon nervous stimuli for their action, the remaining ones dependent on chemical stimuli for their activity. The former would explain the diminution in flow after the injection of atropin. The latter would explain the increase after the injection of choroid or brain extract. Dixon and Halliburton suggest that the chemical stimuli or hormone are some part of the product of the brain's metabolism.

Summary of the Effect of Epinephrin on Cerebrospinal Flow. Table I. The normal secretion for thirty minutes amounted to 5.4 c.c.; or in terms of sixty minutes, 10.8 c.c. Average heart rate, 128 beats per minute. After the injection of adrenalin (1 to 1000) ♏v subcutaneously, the amount secreted in the first thirty minutes amounted to 3.1 c.c., or in terms of one hour, 6.1 c.c., a decrease of 2.3 c.c. in thirty minutes, or in terms of sixty minutes a decrease of 4.7 c.c. Average heart rate for thirty-five minutes after injection of adrenalin was 150.

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[1] Jour. of Physiol., 1913, xlvii.

[2] These secretions dependent upon nervous stimuli are depressed; those dependent upon chemical stimuli are unaffected by the action of atropin.

The following fifty minutes the secretion at the rate of an hour was 15.2 c.c. Three hours after the injection, the blanching upon the arm becoming smaller, the secretion amounted to 14 c.c. per hour. Five hours after the injection the secretion amounted to 13 c.c. per hour. Six hours later the blanching on the arm had entirely disappeared. The secretion gradually diminished and ceased entirely for fifteen minutes, the child apparently being asleep. The child became restless and the secretion began to flow again. The first five minutes amounted to 1.06 c.c. The collection for the following eighty minutes was 5.9 c.c. Seven hours after the injection the secretion collected in sixty minutes amounted to 17.7 c.c.

The chemical analysis of the fluid following the injection of epinephrin shows such marked variations that it would be stretching a point to attempt an interpretation.

We notice, then, that after the injection of epinephrin, the secretion is diminished a little more than one-half in the first thirty minutes. Frazier has likewise reported a marked diminution in his experiments on dogs. What is the explanation? Does epinephrin constrict the cerebral vessels, and by decreasing the blood supply to the choroid plexus diminish the cerebrospinal flow?

The increase in pressure in the circle of Willis after the injection of epinephrin, as is found by Biedl and Reiner, and the decrease in flow from the external jugular vein, reported by Pick, point to a local constriction in the cerebral arterioles. In experiments made upon an isolated brain (in the skull) perfused with an artificial circulation, Wiggers[1] states that the addition of epinephrin causes a diminution in outflow from the organ. He believes the drug constricts the cerebral vessels in the same manner as it does vessels of other organs, and therefore suggests the existence of cerebral vasoconstriction nerves. On the other hand, Bayliss and Hill experimentally could discover no constricting effect of epinephrin on the cerebral vessels. Gerhardt concludes that nothing more than a passive dilatation occurs within the cranium. Dixon and Halliburton, as a result of their experiments on dogs, state that they have never observed constriction of the cerebral vessels from the use of epinephrin, and they challenge the technic as carried out by Wiggers.

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[1] Am. Jour. Physiol., 1905, xiv, p. 452.

Guillard, Huber, and Hunter have demonstrated that the vessels of the brain are provided with perivascular nerve plexuses. But writers as Roy and Sherrington, Hill, Wagner[1] and others from their experimental studies conclude that the cerebral vessels do not possess vasomotor nerves. Masso,[2] as quoted by Howell, has shown by observation upon men with trephine holes in the skull that the constriction of the limbs is always accompanied by dilatation of cerebral vessels. Cushny[3] states that only those vessels of the body that receive nerve fibers from the thoracicolumbar cord are constricted by epinephrin. The blood is, therefore, diverted from the organs supplied by the thoracicolumbar segment, and as a result the cerebral vessels become passively dilated. Suffice it to say, experimental evidence points to the non-constricting action of epinephrin on the cerebral vessels. Further, Frazier[4] has shown experimentally that ligation of both carotids in dogs, thereby decreasing the blood-supply to the choroid plexus has no retarding influence on the rate of the secretion of cerebrospinal fluid. He has also demonstrated that the injection of saline extracts of certain organs, expecially of the liver, kidney, spleen and pancreas, caused a fall in blood-pressure, and at the same time a marked increase in flow of cerebrospinal fluid. Likewise amyl nitrate produced an increase in flow of the fluid. On the other hand, he has shown that the injection of epinephrin caused a rise of blood-pressure, and coincident with this a marked decrease in flow of cerebrospinal fluid. That is to say the general rise in blood-pressure accompanied by an increased flow of blood through the brain, and increased intracranial pressure, is followed by a diminution of cerebrospinal secretion, and the fall in blood-pressure which is accompanied by a decrease in flow of blood through the brain and decreased intracranial pressure results in an increased secretion of cerebrospinal fluid.

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[1] Howell, W. H., Text-book of Physiology, Philadelphia, 1907, p. 580.

[2] Ibid., p. 579.

[3] Text-book of Pharmacology and Therapeutics, Phila., 1910, p. 334.

[4] The Cerebrospinal Fluid as a Problem in Intracranial Surgery, Jour Am. Med. Assn., 1914; lxiii, 287.

It would appear that the cerebrospinal fluid which fills the space of the cranial cavity not occupied by vessels and nerves adjusts the mechanism of the circulation of the brain tissue by equalizing pressure. So nature has adjusted, so to speak, through its nervous mechanism, the secretion of the choroid plexus in such a way, that the activities of the cells are diminished when there is a rise in blood-pressure (increased intracranial pressure) and an increase in the activities occurs when the blood-pressure falls (decreased intracranial pressure.) There is then a certain inverse relationship between the blood-pressure (intracranial pressure) on the one hand and the secretory activities of the choroid plexus on the other, and this relationship has the characteristics of a purposeful adaptation. Therefore, the diminution of flow which occurs after the injection of epinephrin is to be explained by the inverse relationship between blood-pressure and the secretory activity of the choroid plexus.

The fact that a marked increase of secretion occurred forty minutes after the injection of epinephrin is to be explained on the ground that the action of the drug had been spent[1] by this time and as a result the blood-pressure fell with a corresponding increase in flow of cerebrospinal fluid. I was particularly attracted while making my observations and perplexed at the time to find, when collecting my controls, or when urotropin was given, that the secretion invariably decreased when the child was restless, and increased when asleep. At times the patient would be in a violent anger, striking as I would attempt to collect the fluid, and if the hands were held, would toss, scream, and contract violently every muscle. During the entire procedure we were amazed to find the secretion markedly decreased. At other times, after the child had been asleep for some time, restlessness would cause a momentary increase in flow, which is to be explained upon a mechanical basis. However, if the restlessness existed any length of time the momentary increase would be followed by a decrease in flow for the entire time of restlessness. (See Table III.) This likewise carries out the inverse relationship between the blood-pressure which rises during restlessness, and falls during quietude and sleep, and the secretory activity of the choroid plexus, and further substantiates the purposeful adaptation theory. In pathological cases, however, this does not hold good. Here we are dealing with a degenerative process which is taking place in the brain tissue, causing an increase in brain metabolism. Thus there is added to the blood stream a superabundance of hormones, which in turn stimulate those cells of the gland dependent upon a chemical stimulus for their activity to hypersecretion.

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[1] The action of epinephrin when injected intravenously is of short duration. When injected subcutaneously it causes a blanching at the point of injection and in large doses the blanching acts as a reservoir, so to speak, for renewed supply.

We also noticed that there was a marked increase in secretion ten to fifteen minutes after the intake of water; and a very slight change, if any, after the intake of milk. Throughout the entire observations, the pulse rate bore absolutely no relationship to the amount of secretion.

The child was given, on different occasions, large doses of urotropin by mouth, and the cerebrospinal fluid collected in sterile glass tubes at five-minute intervals for several hours after the intake of the drug. We hope within the near future to publish the results of the appearance and disappearance of urotropin in cerebrospinal fluid, as is found in the case of the patient herein reported.

We wish to express our great indebtedness to Dr. Louis Mutschler, under whose service the child was treated, for kindly permitting us to publish full observations of the case.

# THE ASSOCIATION OF SYSTEMIC DISEASE WITH METASTATIC OPHTHALMIA

By G. ORAM RING, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

As is well known, there are certain systemic conditions which by reason of the transference of disease process by way of the blood or lymph channels are capable of inducing violent and wide-spread inflammation of the eye tunics.

A plea for the appreciation of this relationship, its grave significance, the need of coöperation between the general surgeon or physician and the specialist at the first indication of its approach, and finally the possibility of prophylactic treatment in certain bacterial conditions primarily remote from the eye in the endeavor to prevent so serious a complication is the object of this brief communication to an association composed largely of non-specialists.

The lodgement of a septic embolus carried through the blood stream to the capillaries of the retina or choroid with the induction of minute hemorrhages, an inflammatory reaction, the formation of pus and necrotic degeneration rapidly extending to the anterior tunics of the eyeball, this constitutes the usually recognized tragic picture which probably is best called metastatic ophthalmia. The systemic condition which induces it frequently marks it as one of the most fatal but fortunately one of the rarer of ocular diseases.

To quote Alter,[1] the triad of factors necessary for its production are pyemia, embolus, and stoppage. In some cases less violent we have a confined localized abscess formation, with no involvement of the anterior ocular coats, sometimes producing the so-called pseudoglioma. A sacred circle has been drawn around the embolus in recognizing it as the sole explanation by most ophthalmic authorities of the induction of this condition; no other explanation, in any case, being seriously considered, the milder cases only being relegated to the sphere of the toxin.

Bacteria do not act mechanically but by their intracellular toxins, many of which have been isolated, and have produced lesions summarized by Mallory as follows:[2]

The effects which injurious agents, especially toxins secreted by bacteria, produce on the tissues are manifest in four different ways:

By degeneration or necrosis of cells.
By exudation from the bloodvessels.
By proliferation of cells.
By phagocytosis.

He has proved that strong toxins produce degeneration or necrosis of cells with exudation, and weak toxins produce proliferation and phagocytosis; and that the massing of large numbers of bacteria will produce a concentrated toxin capable of causing necrosis and purulent exudation.

The condition under discussion manifests itself as a sequel of many systemic diseases, in addition to surgical and postpartum infection, such as septic endocarditis, influenza, nasal sinusitis with pus formation, parotitis, cystitis, otitis media, synovitis, scarlatina, measles, diphtheria, scorbutus, variola, erysipelas, typhoid fever, pneumonia, anthrax, cerebrospinal meningitis, gonorrhea, so-called Weil's disease, mixed infections, suppurating glands, and at times results from more trivial conditions, such as furuncles and the suppuration attendant upon the extraction of a tooth.

In children, suppuration of the umbilical cord or vaccination may, according to Alter, give rise to pyemia with metastatic ophthalmia. Unfortunately many reported cases give no account of the associated systemic infection.

In rare cases of exanthemata the vision is preserved, and not infrequently in cerebrospinal meningitis, although, as regards the latter condition, we are apt to have an involvement of the eye as an extension by continuity down the vaginal space, rather than a true metastasis.

The organisms responsible for the development of metastatic disease of the choroid are streptococcus, staphylococcus, pneumococcus, meningococcus, pneumobacillus, influenza bacillus, typhoid bacillus, colon bacillus, and mixed infection.

Cheesman and Maltzer[3] found they could cause bacteria to settle in any place they might select by injuring tissue at that place. Flexner and Reed[4] believe focal necrosis due to direct action of toxalbumins, previous injury of the vessel wall allowing transudations to take place.

Pearce[5] believes it evident that while embolism is an important factor in the production, it is not by any means essential in all cases. He regards it as proved that bacteria may find foci of altered or injured tissue favorable to their multiplication.

I mention these views as important, and as indicating that many of these cases can be explained upon the ground of a general bacteriemia; indeed, the viewpoint of Mallory may point to the explanation of why Romberg[6] found only one case of panophthalmitis in forty-two cases of acute malignant ulcerative endocarditis, with the blood containing fibrin masses, bacteria, and disorganized thrombi, conditions that could easily produce emboli, these conditions failing to cause more cases than profound infections distant from the focal lesions.

Axenfeld[7], it will be remembered, proposes the following classification:

Cryptogenic septicopyemia.
Infectious diseases.
Surgical pyemia.
Puerperal pyemia.

The latter two are essentially the same, each being a septic process. It is true that in many instances the bacteria carried through the blood stream to the eye capillaries by their presence or by their evolved toxins induce the destructive inflammation.

Illustrative of the grave possibilities of a furuncle is the case of Augstein[8] (Bromberg) of a strong healthy man, aged twenty-six years, with a furuncle of the neck of twelve days' duration in whom, following a chill, a violent metastatic ophthalmia developed rapidly destructive in character, the exciters of the pus having found their way into the retinal and choroidal capillaries, the bacterial cause being, in all probability, streptococci or staphylococci.

As regards the association of pyorrhea with involvement of the uveal tract, de Schweinitz[9] believes that proper investigation will probably reveal indicanuria, acidosis, and in general terms the usual signs of so-called auto-intoxication, and while treatment to relieve the perverted metabolism may be of value, there is no evidence of a satisfying character that the acidosis, etc., are the cause of the septic mouth condition. It is more likely that oral sepsis originates the auto-infection as well as the local inflammatory processes in joints and uveal tract disease.

An increasing experience in the study of metastatic choroiditis resulting from puerperal pyemia will probably show that the ocular disturbance is due rather to the general bacteriemia than to a specific lodgement of embolus.

So-called postpartum ophthalmitis usually occurs about the sixth day after delivery and rarely after the end of the second week. It is either unilateral or bilateral, the latter being essentially fatal in from 85 to 90 per cent. The unilateral cases give much better results as regards life, but the eye involved is usually lost.

de Schweinitz[10] reports a recovery from puerperal pyemia associated with a bilateral metastatic ophthalmia; in this case, however, there were ring abscesses in each cornea followed by panophthalmitis.

Two monocular cases are reported in Norris and Oliver's[11] *System* as occurring after normal confinement in perfectly healthy young women. The disease progressed to a point of producing large, rounded, bluish-white, flocculent masses like packed cotton-wool. Neither showed signs of external inflammation. Such a condition was not likely to be due to a septic embolus, but possibly to a metastatic disorder of intestinal origin.

In the conditions we have been discussing, violent poisons are present and circulating in the bloodvessel system, and must be neutralized by every known process. Hexamethylenamine and helmatol are excreted in the anterior chamber in about three hours. The excretion is increased by the use of a mydriatic locally, and while according to Gradle[12] the concentration in which they occur in the anterior chamber will not inhibit the growth of virulent organisms in the test-tube, yet it is believed it will weaken them and allow them to be more easily killed by the normal antibodies in the various secretions. In addition to general measures directed to the gastro-intestinal tract, kidneys, skin, and circulation, hypodermoclysis being of recognized value in lessening the concentration of the toxins, vaccine and serum therapy are of distinct importance, and should be so recognized and exhaustively tried, although their exact value has not been definitely established.

One difficulty in their use, from the view-point of the eye surgeon, is the length of time required to ascertain the offending organisms after the eye is involved before serum or vaccine treatment can be instituted.

It is recognized that, whenever possible, an autogenous vaccine should be given precedence over a stock vaccine. Let a smear be taken from the focus of infection and the result should be known in thirty minutes.

Then let the stock sera or vaccines be given at once upon making a definite diagnosis, and, at the same time, have prepared an autogenous vaccine for use when a second dosage is indicated.

In the matter of the coli vaccines it is well known that the most distinguished authorities decry the use of any but autogenous ones. It must be remembered that there are probably from twenty-five to thirty varieties of this bacillus which are not to be distinguished morphologically; therefore we must, unfortunately, wait until an autogenous culture can be prepared or utilize the polyvalent variety which may be weak in the very organism that is inducing the serious infection. This, it seems to me, may explain why the stock vaccine of this series of organisms has been of so little use. If it is true that the majority of cases of choroiditis are due to a primary bacteriemia, producing a local cellular degeneration or necrosis, fluid extravasations, weakened arterial walls, hemorrhages, the local invasion of leukocytes and exudation, with, finally, abscess, and that emboli are less frequently the active factor; if the condition of the eyes can be an indication of the virulence of many cases of infection, then there should be, if necessary, repeated examination of the eyes as an aid to diagnosis as pointing to the virulent character of the infection and as an indication for more active systemic treatment. It is, of course, regretfully recognized that up to the present we have no absolute specific in the more rapid and virulent types of the disease. In such cases, however, particularly those of the streptococcic variety, large doses of the serum should certainly be tried.

Mayou[13] suggests that in streptococcic infections of the eyes polyvalent serum treatment is most useful, and that treatment should be instituted early.

As regards the use of vaccines in the treatment of intrinsic disease of the eye, Bryon emphasizes the importance of using larger doses, and thinks that when so used there is less danger of the production of the negative phase. When we are dealing with a condition due to a pyogenic organism it is best, as insisted upon by the same authority, to use a specific vaccine from the focus of infection (autogenous); but in a disease in which staphylococci occupy a causative relation, stock vaccines are efficent.

He reports fair success in using the pneumococcic stock vaccines, giving, as soon as the diagnosis is made, 25,000,000 stock pneumococci vaccine. As pointing to a promising field for vaccine treatment, I wish to refer to a case reported by Bently,[14] of Seattle, of metastatic ophthalmia due to colon bacillus, in which the usual tragic denouement of a panophthalmitis resulted; and in connection with it, to bring to your attention two cases of metastatic inflammation associated with a colon bacillus toxemia, by Mr. Arnold Lawson,[15] of London. The latter did not proceed to a panophthalmitis, but one was optic neuritis and the other was an infiltration of the cornea with extensive exudate. On the fifth day of primary infection a mixed vaccine of Streptococcus, bacillus catarrhalis and gonococcus was tried without effect. The colon bacillus was present in the urine in vast numbers.

A vaccine of the Bacillus coli was injected on the ninth day; the conjunctival discharge rapidly decreased and the cornea cleared. Five injections were give: the first 5,000,000 and the last two each 100,000,000. The bacteriemia rapidly improved after the first injection.

As pointing to the frequency of puerperal sepsis the value of sera and vaccines in its treatment, and consequently of the possible prophylactic value of such treatment in the prevention of metastatic ophthalmia, a personal communication was addressed to a number of distinguished specialists in obstetrics and gynecology of this city. The valued replies are herewith presented:

Dr. E. E. Montgomery, professor of gynecology in Jefferson Medical College, advises me that he has never seen a case of metastatic ophthalmia as a sequel of puerperal sepsis, but adds that in such a case he would immediately use anti-streptococci serum until vaccine could be secured, and expresses great confidence in both the sera and vaccines.

Dr. Barton Cooke Hirst, professor of obstetrics at the university of Pennsylvania, has seen but one case of metastatic ophthalmia as a sequel of puerperal sepsis. In that case the eye was lost. Dr. Hirst states, however, that he has used the antisera very extensively in puerperal infection, occasionally with excellent results, although not invariably. In his experience the vaccines are not so successful except in subacute inflammation of the large joints in which case he has seen vaccines give brilliant results.

Dr. Richard C. Norris, surgeon in charge of the Preston Retreat, advises me that in 8000 consecutive obstetric cases there is no record of a case of metastatic ophthalmia, and that he has never had a case under his care in private or hospital practice. As Dr. Norris suggests, this negative report may be of value, and is in my judgment, an eloquent tribute to modern methods as exercised by these masters in the practice of obstetrics.

Dr. William E. Parke, consulting obstetrician to the Protestant Episcopal Hospital, has not seen a case of the disease in question as a sequel of puerperal sepsis, but has used an autogenous vaccine in one case of sepsis without eye involvement, but without being impressed with its value, the patient having died after one hundred and fourteen days' illness. It is quite possible, however, that the treatment may have prolonged the life of the patient.

Dr. J. A. McGlinn writes that he has seen a large number of septic cases, both after abortion and labor at term, but has had none with metastatic eye infections. On the theoretical ground he regards serum as proper treatment, but in the dose usually advised thinks serums do little good. He uses serums and vaccines only occasionally, and thinks the large dosage advised by Hirst makes the cost to the ordinary patient prohibitive.

Dr. J. Osborn Polak, professor of obstetrics in the Long Island Medical College, is quoted by Dr. McGlinn as a strong advocate of vaccine treatment, an enthusiasm he does not share. McGlinn further states that some men even believe that it is bad practice to empty the uterus in the presence of infection though it contains some of the products of conception. He advises the general upbuilding of the patient and the avoidance of anything, even surgery, that will lower vitality. His treatment further consists of stimulation when needed, forced feeding, measures to combat high temperature, ice to relieve pain, and having the patient in bed in the open.

By reason of the fact that the infection from puerperal sepsis is so difficult to combat, I felt it wise to give in some detail the opinion of these distinguished representatives of this branch of professional work.

Rigid antisepsis in surgery and obstetrics is eliminating much of this form of disease; but if the general physician, surgeon, or obstetrician into whose hands these urgent cases of septic disease may fall will immediately have them studied from the bacterial view-point, and promptly institute serum and vaccine therapy in addition to the most approved general methods (as for instance those outlined by McGlinn in the puerperal forms of disease), aside from the value of such treatment as influencing the systemic infection, it is quite possible that the general regime plus the special therapy may act in many cases as a prophylactic against transmission of the poison to the ocular tunics. Assuming that such treatment will be utilized, we believe that it is not unlikely that the opportunity afforded the ophthalmic surgeon of seeing cases of metastatic choroidoretinal disease may be still further reduced.

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# CONCERNING COLOBOMA OF THE IRIS IN ASSOCIATION WITH CONGENITAL CATARACT

By G. ORAM RING, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

THE extreme difficulty and in many instances the absolute impossibility of adequately solving the etiological problem in certain faults in ocular development is illustrated in the pronounced defects in the cases of the mother and two children in the one family herein reported, and it is suggested that there may be an association between a vicious prenatal environment and the development of the ocular defect. In a hospital service of about 3000 new cases yearly such cases have been rarely met with.

In the case of the mother and child now shown, each with definite abnormalities of cornea, iris and lens, and with a history of a second child of the same parents studied by me at an earlier period but now deceased, the element of heredity is strikingly evidenced.

The patient, Mrs. T. R., the mother of four children, two of whom were born with ocular abnormalities, shows the following condition:

O. D. The iris below is notched or slightly folded upon itself and the pupil when dilated alters the position but slightly. At the posterior lens pole is a dense white opacity. Nuclear riders are present and numerous dots of extensive denseness suggestive of calcareous change are scattered throughout the largely opaque lens.

O. S. There is a coloboma of the iris below, with similar irregular lens opacity.

Microcorneæ are present in each.

In the child exhibited, microphthalmos is present in each eye.

In the posterior cortex of the lens in O. D., is an irregularly round calcareous-like nodule, and the area of the nucleus is marked by a number of riders and several small dot-like opacities.

O. S. Same character of nodule, nuclear area is present but less distinct, and calcareous-like dots absent.

In both eyes the iris below shows a concave depression corresponding in location to the coloboma of the mother.

The second child with a congenital defect died at five months, but had microphthalmos, double coloboma, and lenticular opacities similar to those of the mother and child shown.

The rather vicious prenatal history of this little mother is of interest.

On the paternal side the father was a confirmed alcoholic and user of tobacco, now dead. Cause of death and age at time of death unknown.

Mother is living, now aged sixty-two years, who during her married life was compelled to submit to exceedingly harsh treatment, having passed through periods during which she received insufficient food and at times lacked even shelter. Gave birth to eight children—four boys and four girls—at periods of about twenty-two months, the patient being the fourth in order; previous to the birth of the patient the ill-treatment accorded her mother was particularly wretched.

The patient, Mrs. T. R., was born in England, and was a victim of poor health during the first two years of her life. At the time of immigration to this country, owing to her sickly condition she was at first refused passage. Teething occurred very late, that is at thirteen months. During her childhood she passed through measles and a very severe attack of typhoid fever, but from twelve years of age and onward she has had very little physical discomfort.

She is the mother of four children: the first a girl, died at seven months of gastro-enteritis; the second, one of the patients shown; the third a child dying at five months who presented the congenital defect referred to, and a fourth child, now aged four months, who shows no lesions whatever in either iris, choroid, or lens.

Congenital abnormalities of the iris in the well-known classification of Lang and Collins consist in:

1. Variations of color.
2. Persistent pupillary membrane.

3. Tremulous iris (iridonesis).

4. Deficiency of the whole iris (iriderernia).

5. Deficiency of a portion of iris structure (coloboma). With the well-known associations of coloboma of the iris (choroidal and retinal coloboma, microphthalmos, coloboma, and displacement of the lens, coloboma of the nerve, and cataract) we are familiar.

It is curious to note that a number of the text-books fail to mention the association of congenital cataract with the iris defect. This combined defect de Schweinitz especially mentions and likewise draws attention to the more frequent involvement of the left eye.

Fuchs refers to the frequency with which cataract develops with coloboma of the choroid and regards the cataract as usually complicated and inoperable.

Let me recall to your memory that previous to the formation of the iris, the anterior segment of the eyeball consists of the cornea, anterior fibrovascular sheath, lens and capsule, and posterior fibrovascular sheath and ciliary body; that the anterior fibrovascular sheath receives its blood supply partly from the ciliary arteries and partly from the vessels of the posterior fibrovascular sheath which are prolonged around the sides of the lens to join it.

The iris is developed by growing forward from the margin of the anterior chamber, and in so doing insinuates itself between the cornea and anterior fibrovascular sheath on the one hand and the lens on the other, pushing the prolongation from the posterior fibrovascular sheath in front of it. The anterior fibrovascular sheath becomes the pupillary membrane.

If we suppose, as recorded by Swanzy, some abnormal adhesion to occur between the cornea, anterior fibrovascular sheath, and lens capsule; or some delay in their separation at the whole circumference of the future anterior chamber, we can understand how a mechanical obstruction to any growth of the iris forward would be induced.

Coloboma of the iris and its complete absence have been shown by Treacher Collins to be different degrees of one and the same condition.

The object of this brief communication is to recite a rather interesting history and to learn whether or not your opinion accords with my own, namely, that in view of the increasing haze of each lens I feel warranted in and propose doing a careful series of discissions, beginning with a vertical incision through capsule into lens extending from the lower to the upper margin of the dilated pupil.

NOTE.—In the discussion which followed the reading of the above before the Ophthalmic Section of the College of Physicians of Philadelphia, December 1915, the consensus of opinion favored the procedure proposed.

# CONCERNING SUDDEN OBSTRUCTION TO THE RETINAL CIRCULATION IN A CASE OF CARDIO-RENAL DISEASE

By G. ORAM RING, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

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J. C., male, aged fifty-seven years, was referred to my sevice at the Episcopal Hospital, from the medical clinic of Dr. R. S. Hooker.

*Family History.* Father died of heart disease, mother and two brothers of unknown cause. One brother and two sisters living and well.

*Previous History.* Patient is a boilermaker by trade. Denies ever having been ill until two years ago. A regular user of tobacco since eleven years of age. Has been addicted to the use of alcohol in form of whiskey and beer from nineteen years of age until seven years ago, since which time he has been strictly temperate. He drank regularly and usually to excess on Saturday and Sunday. Denies venereal disease, and comes for treatment on account of palpitation and dyspnea.

*Present Illness.* Twenty-seven months ago while sitting at his work he found it impossible to elevate his arm above the shoulder. The attempt to do so was accompanied by slight circular movements of the hand. Upon attempting to rise an attack of vertigo came on accompanied by a sensation of pins and needles pricking his left arm, which was improved by rubbing. He immediately walked to a physician's office five blocks away and being sent to his home by automobile, found he could not get out of the machine unassisted neither could he walk. Hemiplegia lasted but a brief time, and within three days he was able to walk with the aid of a cane which he used for several weeks. He is fairly nourished, has a hemiplegic gait upon the left side and holds arm in characteristic hemiplegic position.

Left arm and leg spastic. Reflexes: knee-jerks much increased on left; Babinski present; also ankle clonus on left side.

Heart enlarged to left. Mitral systolic murmur. Accentuated aortic second sound.

Urine shows specific gravity 1017; faint albumin ring, with hyaline and granular casts; no sugar.

Wassermann: negative.

Blood-pressure: September 14, 1915: systolic, 250; diastolic, 130. October 1: systolic, 220; diastolic, 120.

During July, 1914, one afternoon at four o'clock vision in left eye suddenly became foggy, and was reduced to distinguishing forms, but returned on following morning.

October 21, 1915. Had temporary loss of vision in right eye similar to that recorded in O. S. in July, with restoration of sight the next morning. Four days later he awoke to find vision in right eye gone and two days later was seen at the eye department of the Episcopal Hospital.

October 27. O. D. Neither light perception nor projection was present. Pupil semi-dilated and immobile to direct light stimulus but maintained a consensual reaction. The fundus was markedly edematous, some of the arteries being visible as slender cords, veins very small. The macular region shows markedly red through the haze, contrasting with the white of the remaining fundus and picturing the so-called embolism of the central artery.

O. S. Media clear, nerve of good color. The superior nasal artery just beyond the disk is contracted and the connective-tissue increase is plainly visible. The superior vein shows an indentation at the point of crossing. In the macular region above is a patch of infiltration bordered with a hemorrhage. The immediate area of the macula is infiltrated, and a disk diameter to temporal side is a flame-shaped hemorrhage. Entire fundus is diffusely hazy.

The following day the superior artery was seen as a white cord. The circulation terminating after its emergence from the disk centre at a point about midway between the point of emergence and the upper disk border.

The inferior artery is obstructed at about the same point, and the artery traced as a white cord into the periphery. Between the nerve and macula the edema is marked, no details possible. The nerve is covered with dilated and slightly tortuous cilio-retinal vessels.

Twenty-four hours later the systolic blood-pressure had fallen to 200, the diastolic to 130. Pulse pressure therefore 70 and the following changes were noted:

The superior artery in O. D. now shows as a series of broken red lines and the inferior artery runs into an edematous area below similar broken red lines.

On October 31, the superior artery is manifest as a continuous red line somewhat broader and the inferior artery is filled both before and after leaving the edematous area. The artery to the nasal side is constricted at the point of emergence from the disk.

On November 2, both superior and inferior arteries were filled, the superior being contracted after leaving the disk and appearing as a thin red line.

The edema between the disk and the macula is clearing, a hemorrhage borders the vein, area of infiltration is evident in macular region with a deposit of cholesterine.

The latest note on November 16, in O. D., shows that the superior artery, after leaving the constriction which is above the nerve, runs into an edematous area and is lost in spots. At the point of crossing it indents the vein. The superior temporal branch is lost to a point near the macular region beyond which the terminal branches can be seen. The inferior temporal branch is constricted and a hemorrhage adjoins it in the macular region. Inferior artery runs into an area of edema and organized inflammatory tissue. Inferior nasal branch cannot be seen after leaving the nerve.

O. S. Superior temporal artery is constricted at nerve border with lipoid infiltrations along its course and with similar deposits in the macular region.

Superior nasal branch interrupted in its course, retina edematous.

Blood-pressure: Systolic, 190; diastolic, 110; pulse pressure, 80.

The phenomenon above described, particularly the condition noted at the first visit with the cherry-red spot at the macula, pointed to what is commonly known as embolism of the central artery. A number of possibilities of explanation of this red spot have been made: little hemorrhages, retinochoroiditis, yellow pigment of the macula, but that most usually now considered is that it is caused by the red of the choroid shining through the retina at the point where it is thin and the brilliancy is accentuated by a contrast of its surroundings, the cerebral layer of the retina in which the cloudiness is situated being absent at the macula.

By a true embolism we mean an occlusion produced by a plug driven into the artery. It is now believed that this condition is extremely rare and that the changes noted probably result from thrombotic processes in the central artery excited by the degenerative changes in the walls. In other words that we are probably dealing with a proliferating endarteritis, in which the lumen is contracted by a progressive proliferation of the intima.

The restoration of the circulation is made possible by a retrogression of this process.

According to Kern no cause for an embolus can be found in 66.3 per cent. of cases, so that a local disease in the artery is probable in the vast majority.

We are able to diagnose an endarteritis rather than an embolus when we are able to see the walls of the diseased vessels either just after the occlusion or at a later time.

According to the researches of Harms the diagnosis of a true embolism is justified only when we can exclude a primary disease of the wall, when a defect of the valves of the heart or some other plausible condition of disease can be demonstrated as its probable cause, and when signs indicative of a thrombotic occlusion of the artery are not present.

Arteriosclerosis is characterized by the occurrence of changes in and thickening of the intima which was supposed by Rokitansky to be due to the depositing of cells directly from the blood-stream, by Virchow to be a true inflammatory hyperplasia as the result of some so-called formative stimulus, and by Thoma to be a compensatory thickening of the wall in order to diminish the lumen of the vessel after the stretching which occurred under the increased blood-pressure with which it was usually associated. Jores, on the other hand, regards this as a true hyperplasia resulting from the high blood-pressure, but independent of the lumen of the vessel. Emphasis has here been placed upon the intima with no reference to the important inflammatory changes in the *media*.

Koster lays special emphasis upon the importance of the degenerative and calcareous changes of the intima and adventitia as well, and believes the process is an infiltration in the adventitia surrounding the vasa vasorum like a sleeve and following the vasa vasorum into the media, and that there is almost always a demonstrable continuity between the patches of endarteritis, mesarteritis and periarteritis.

The vasomotor crisis of Pal, with sudden constriction and dilatation of the arteries in association with the organic changes in the vessel walls, furnishes a basis for the interesting changes noted in the comparatively prompt reëstablishing of the blood-current in vessels that a few hours previously had the appearance of white cords.

NOTE.—I am indebted to Dr. Hooker for his notes regarding the systemic examination of the case, and to Dr. W. F. Hopkin, of my dispensary staff, for his care in recording changes which were the outcome of alterations of pressure.

# PENETRATING WOUND OF CORNEA, IRIS AND LENS; NEGATIVE X-RAY EXAMINATION; OPERATIVE RESULT[1]

By G. ORAM RING, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

The pointing of the clinical picture in the case history herewith presented, toward a probably entrenched foreign body, the definite and urgent advice previously received from a specialist of repute to submit to an immediate enucleation to prevent loss of the fellow eye, the importance of the negative *x*-ray finding as furnishing basis for conservative viewpoint and treatment, and finally the ordinary operative intervention with subsidence of all irritation and the attainment of approximately normal vision under appropriate correcting lenses, constitute an excuse for recording a brief note of personal interest.

J. A. was first seen January 23, 1915, when the following history was elicited:

While turning steel mandrels on a lathe during March, 1914, he was struck in the right eye, presumably by a small piece of steel, after which the eye promptly grew red and painful, the inflammatory symptoms subsiding after about two weeks.

The vision gradually grew dim and progressed until at the period of his visit to my office about ten months later, only light perception and projection were present. No photophobia of the left eye was evident but some lacrimation which recurred at intervals but which has now subsided.

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[1] Read before the Ophthalmological Section of the College of Physicians of Philadelphia, November, 1915.

Seven months after the accident he was strongly urged to submit to an immediate enucleation.

Upon the date of my first examination the following condition obtained:

In O. D. a definite zone of pericorneal flush was present down and in, which was immediately accentuated and became general under examination and oblique illumination and was combined with some lacrimation. On the lower and inner zone of the cornea, just below the horizontal diameter, a small scar was present, and by careful examination a minute slit-like perforation of the iris in and slightly up close to the inner pupillary margin and a shade over a millimeter in extent, extending inward could be noted and through this area the faint glimpse of opaque lens could be detected. The lens was cataractous of the frank gray type with a few crevices in the pupillary area marking the only points where it lacked completeness of opacity. The tension was about normal and the light perception and projection were good in all parts of the field. The pupil in O. D. reacted promptly to light stimulus and no adhesions were present. Dilatation under mydriatic was only moderate.

In O. S. vision was 6/5, no gross changes in the ocular fundus being present. The upper, inner and lower disk margins were somewhat obscured but no lacrimation nor undue sensitiveness to light were evident.

50 m. print was read from 20 to 50 c.m. Vision with correcting glass $-.75$c. ax. $80° = 5/4$. The patient was immediately referred to Dr. Wm. M. Sweet, who reported that no foreign body could be located after two careful trials, notwithstanding the fact that the clinical picture strongly indicated its presence.

The patient was seen at intervals for several months and on May 25th consented to operation which was done at the Episcopal Hospital: A vertical incision extending from the lower to the upper dilated pupillary border was made in the lens capsule with a Hays-Ziegler knife. A second incision was made on June 4. The lens swelling and subsequent absorption were wholly satisfactory and without pain or redness. Finally the V-shaped incision in a faint remaining posterior capsule on November 4 made possible a vision almost normal.

The *x*-rays will not in every case locate a foreign body, but the failures are so very rare in the presence of a skilled technique that one is usually warranted in basing an operative judgment upon its findings.

During the period through which the above case was under observation, two other perforating foreign body cases, each with traumatic cataracts, came under my care, one negative to *x*-ray examination, the other positive. The former was treated in a precisely similar manner to the case outlined, but here the perforation was central and the iris uninvolved. In the latter the foreign body in the lens was extracted with the lens substance, but in this instance a traumatic infected ulcer with posterior adhesions was present when the case came under observation. The healing in each case was uneventful, but final discissions of the posterior capsule of the latter two cases remain to be done.

# UNILATERAL PULSATING EXOPHTHALMOS[1]

By G. ORAM RING, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

The object of this brief communication is to place upon record a case of pulsating exophthalmos, in which, based upon the history given by the mother of the patient, it was thought possible to associate the etiology with a congenital growth which manifested itself at the upper and inner orbital margin, which was operated upon when the patient was two years of age, and was followed later by a second operation, which in turn was followed by a vascular orbital distention evidently beginning in the region of the caruncle and the upper and inner orbital region which induced a pronounced and slowly progressive proptosis forward, downward and outward.

The disease has extended over a period of approximately thirty years, with a slightly functioning eyeball and with the possibility of lid closure until about one month ago, when an acute influenzal attack accentuated the condition, changing a white to a markedly red eyeball with the induction of an exposure keratitis involving the lower third of the cornea.

Miss H. H., white, aged thirty-two years, was admitted to the medical ward of the Episcopal Hospital, December 26, 1915, in the service of Dr. A. A. Stevens, and upon recovery from an acute systemic influenzal infection was transferred upon January 4, of the present year to my eye service.

Her mother is living at seventy years of age. Father said to have died of thrombosis at sixty-eight, and there is reported to be a history of cardiorenal disease of each parent.

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[1] Read before the Ophthalmological Section of the College of Physicians of Philadelphia, January, 1916.

Patient has had no previous illness other than measles and pertussis, but was a rather delicate child.

She was born with a small tumor on the left upper eyelid above the inner canthus, which showed a disposition to increase in size as she approached two years of age, at which time it is said to have been removed by the late Professor William F. Norris, at the University Hospital on May 1, 1883.

The operation was followed some months later by a second, involving the region of the caruncle. Gradually the orbital tissues began to enlarge until a condition presently to be described was in evidence upon the patient's admission to the hospital.

Through the courtesy of Doctors Holloway and Langdon a brief dispensary record from the Hospital of the University of Pennsylvania, of May 1, 1883, has been given me, and is as follows:

Hortense H., aged two years, May 1, 1883. House case. Admitted to hospital for operation. Diagnosis: Cyst of orbit. Enlargement of caruncle.

The child has growing from the inner caruncle of O. S. a third lid, and also a cyst at the upper and inner angle of the orbit, deeply seated. History of suppuration, but no outlet found.

May 3, 1883. Cyst and third eyelid removed. Ether. (Operation by Prof. Norris.)

Since the subsequent vascular growth apparently developed from the region of operative intervention it is unfortunate that the results of subsequent studies are not obtainable as the case has been without professional advice until the recent acute attack produced pain and ulceration.

The patient upon admission to the Episcopal Hospital had the ususal signs of a so-called grippe infection, with a beginning pneumonia, in the stage of congestion of the lung tissue of the right base.

The inception of her attack nine days before admission was marked by a chilly sensation up and down the spine, cough, headache and general muscle ache, with expectoration of some blood and with moderate epistaxis.

Three days after admission she presented catarrhal symptoms of the eyes, nose and bronchial system. Temperature, 102.3° F.; respiration, 28, with a productive cough and impairment of the lung note over the right base posteriorly and with increase in tactile fremitus; a pneumonia in the stage of congestion was diagnosed. By January 2, patient presented few subjective symtoms, but complained of pain in O. S., with a serous discharge, an injected eyeball and corneal ulceration.

Examination upon admission to the eye ward is as follows: The left eyeball is proptosed forward and downward and outward, the pole of the cornea being 17 mm. farther forward than the normal or fellow eye and 10 mm. below it.

The eyeball is greatly injected, its movements moderately well preserved, the cornea at its lower third presents an ulcerated area of exposure keratitis, the upper border corresponding to the point of closure of the lid. Pupil is somewhat dilated and at the inner angle shows dilated portion of circular vein.

In the lens up and out is a triangular dense subcapsular opacity from the bottom of which a spur passes into the lens substance. The edges of the opacity are eroded.

The fundus which is difficult to examine shows areas of retinochoroiditis with extensive choroidal atrophy. The veins of the fundus show only moderate dilatation.

The forward swelling and distention of the lid area begins 10 mm. above the brow and the measurement from this point to the edge of the lid is 40 mm. The veins of both lids are markedly dilated and the distance to the terminal of the swelling and edema of the lower lid from the point of beginning in the brow is 58 mm. Under the upper lid is an enormous blue-red swelling with a thickened fold of conjunctiva, growing less toward the outer canthus. A similar less marked condition of the conjunctiva and vascular tissues exists beneath, with a symblepharon at about the inner third.

There is an area of ecchymosis on the outer third of the lower lid, a serosanguineous fluid is more or less constantly oozing from the conjunctival vessels, and over the lid area a bruit can be distinctly heard.

In a good light fingers can be indistinctly outlined.

Since there is no history of traumatism other than that induced by the original operative intervention, the case is regarded as an aneurysmal dilatation of one of the branches of the internal carotid after it leaves the sphenoidal opening, probably secondary to the original operative procedure.

As is well known, pulsating exophthalmos may be an aneurysm of the ophthalmic artery either intra- or extra-orbital, an aneurysm of the internal carotid or a so-called arteriovenous aneurysm involving both carotid and the cavernous sinus. Dilatation from the obstruction of ophthalmic vein is also frequently in evidence. It is not always possible to differentiate these different conditions nor is it absolutely necessary from the viewpoint of the treatment if ligation of the carotid is decided upon.

Note.—In the discussion which followed the reading of the above clinical history it was suggested that 2 per cent. solution of gelatin in normal saline solution be used by subcutaneous injection every third day, the dose being 20 c.c. This was tried for two months, at the end of which time ligation was decided upon.

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# A CASE OF PULSATING EXOPHTHALMOS, NON-TRAUMATIC, DUE TO ANEURYSM

By FREDERICK KRAUSS, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

On June 17, 1915, Mrs. Margaret B., aged fifty-seven years, was referred to me by Dr. Seiffert on account of swelling of her eyes. She complained of inability to lift her right eyelid, blurred vision, and a continuous noise in her head, especially on the right side.

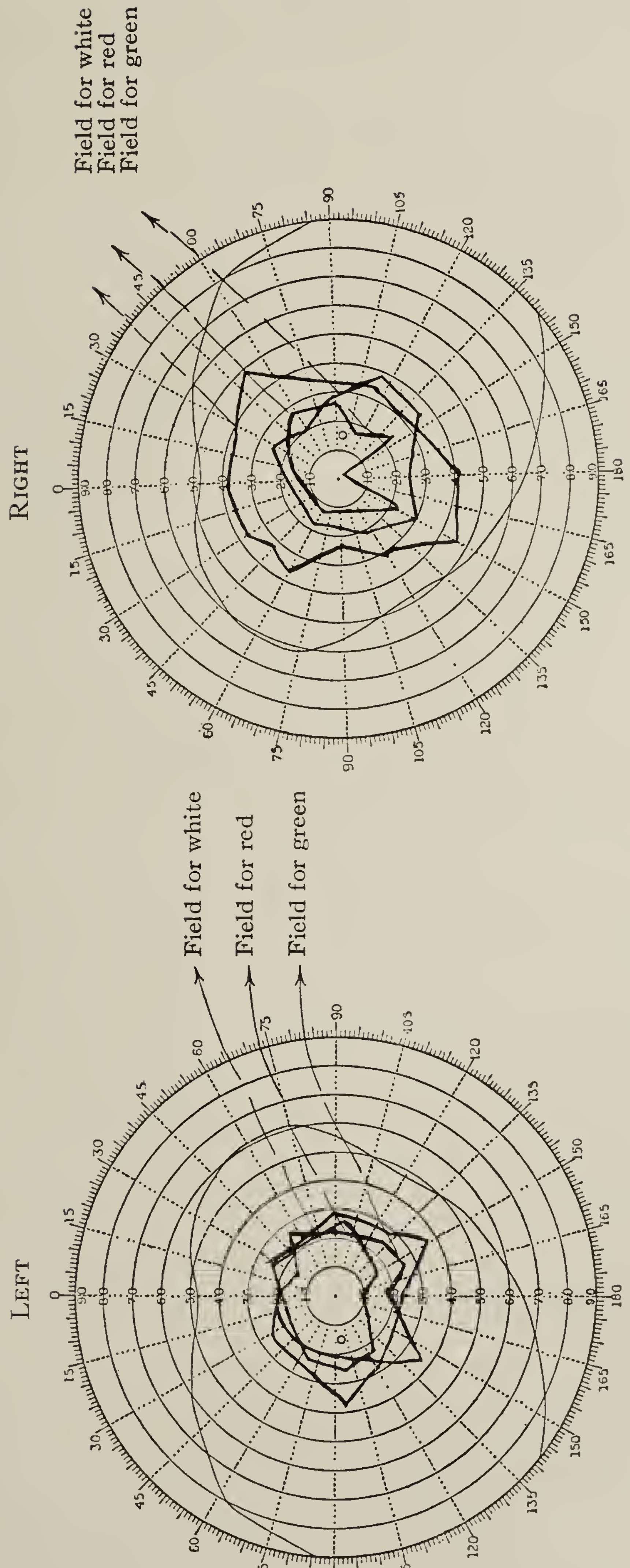
The ocular condition when first seen was as follows: Both eyes were proptosed, especially the right eye, which was very prominent. The lids were dusky, the conjunctiva greatly congested and edematous, hanging in folds beyond the lid margins, completely shutting off the sight from the right eye, the upper lid of which was immovable. The vision in the right eye was 5/22.5 and in the left eye 5/xv. Ocular movements were restricted especially up and out. The fields of vision were greatly and concentrically cut in both eyes, with slight crossing of the color fields (Figs. 115, 116).

The left eye was freely movable; the exophthalmos, though very pronounced, was less than in the right eye. The pupils reacted to light and accommodation.

In the right eye the ophthalmoscope revealed deeply filled tortuous veins, the arteries being somewhat contracted, with no hemorrhages.

In the left eye the nerve was gray in tint, the vessels full, but there was no swelling of nerve or retina. There was no pulsation in either eyeball, but there was a very loud bruit accentuated with every cardiac impulse, heard all over the face and head, but loudest over the right temporal bone anteriorly.

The following history was obtained by my resident, Dr. Belk, upon her admission to the wards of the Episcopal Hospital, June 21, 1915:



Figs. 115 and 116. M. B., No. 382. No scotoma.

The patient has three children, living and well. One child died of marasmus. One miscarriage. No tuberculous or hereditary disease.

*Personal History.* Rheumatism and some palpitation of the heart for several years off and on. Had usual diseases of childhood. Drinks coffee to excess.

*Present Illness.* Began about six weeks ago, with throbbing on top of her head, and severe continuous headaches, gradually getting worse. There is no history of traumatism.

Six weeks ago the right eye became bloodshot and painful, followed four weeks later by a similar condition of the left eye. At the present time she has continuous throbbing and aching in the eye and forehead, photophobia and lacrimation and impairment of vision in the right eye.

*Physical Examination.* The following notes were dictated by Dr. E. J. Morris of the Medical Staff:

She has severe palpitation but no dyspnea or cardiac pain. Has vertigo and tinnitus. Constipated habit. She has no symptoms referable to the pulmonary or nervous systems.

She shows bad teeth, tonsils enlarged, pharynx injected. Lungs showed a few vesicular rales, at both bases posteriorly. At the right apex posteriorly is a small area of prolonged expiration.

Heart outline: Right border of sternum, upper border at the third rib, 6 cm. to the left of the midclavicular line. The sounds are regular and of good muscle quality.

Over the aortic area is heard a soft double murmur, occurring before and after the second sound. The aortic second sound is accentuated.

The pulses are equal and synchronous. The head nods with each heart beat.

Abdomen is large. No masses or tender areas or fluid.

Extremities: Slight edema of ankles. The knee-jerks are absent on both sides. Station is fair. No Romberg sign.

The *x*-ray showed nothing abnormal except a cloudiness of the orbits. The Wassermann test was made on June 24, August 23, and September 15, 1915, being uniformly negative.

The spinal fluid showed: Cell count, 5 per cent.; Nonne, negative; Noguchi, negative; Fehling's, negative.

At a later test similar findings were noted except that Fehling's was reduced. The blood count on June 21, 1915, was 8240, which increased to 12,320 on October 2, 1915, with 41 per cent. polymorphonuclears.

The blood-pressure was 202 systolic, 125 diastolic. The patient was put to bed on light diet, keeping bowels and kidneys free. She was given increasing doses of potassium iodide.

Dr. Thomas R. Neilson examined the patient for me, with a view of tying the internal or common carotid, but on account of the high blood-pressure and mental sluggishness of the patient, was disinclined to assume the risk.

Upon explaining the danger of the operation to the patient and her friends, operative interference was declined.

Several weeks passed under this expectant treatment, during which both eyeballs bulged out enormously, being hidden by tremendous folds of chemotic conjunctiva arising from the fornices. The right eyeball became fixed immovably, and apparently held by the tension of the orbit at congestion which was light bluish in color, therefore apparently venous and serous, the latter evident in the conjunctiva. Attempts were made to relieve the chemosis by scarification, which was partly successful. In the left eye, the ocular movements were greatly diminished but not abolished.

There was free conjunctival secretion. At this time argyrol and hot compresses were used in conjunction with cleansing.

The vision became worse, gradually dropping to counting of fingers. Upon receiving the refusal of the patient to be operated upon, the resident and nurse were instructed to compress the common carotid artery on the right side against the vertebra until the lumen was completely occluded. This was readily ascertained by the absence of pulsation in the facial artery and by the cessation of the loud bruit in the skull. At this time there was no pulsation in either eye.

Following this there was a gradual diminution of the size of the exophthalmos, until it was normal in the left eye and but slight in the right. The patient complained but little of the noise and throbbing and the bruit was much fainter. This was on August 24, 1915.

The following day the patient experienced great pain in the right eye, with much chemosis of the lids, with recurrence of the bruit and a marked pulsation of the right eye. The left eye was also involved, but to much less degree, with no pulsation. The continuous blowing murmur was now most pronounced over the right eyeball, and slightly over the temporal bone.

The right eye gradually became fixed with apparent palsy of all of its muscles, including the sphincter iridis which was fully dilated and immovable. There was complete ptosis. The bulbar conjunctival vessels were deeply filled with passive congestion, the conjunctival sac in the fornices filled with serous exudate and extending in huge folds between the upper and lower lids and eyeball.

The eye-ground showed great diffuse edema of the optic nerve and retina, with deeply swollen veins seen at intervals. The swelling was less pronounced in the periphery. There were many retinal hemorrhages in addition to which there were numerous radiating streaks of hemorrhagic tint. These were probably enormously engorged choroidal vessels. The vision was reduced to counting fingers at 9 inches.

A few weeks later the hemorrhage had been partly absorbed, the retinal edema greatly diminished, the arteries much contracted, the superior temporal veins very full and tortuous.

On October 18, 1915, she developed a low grade of iritis, with slight pericorneal injection, causing posterior synechia, descemetitis, and haze of the vitreous.

The eye became absolutely blind. Under continuous treatment consisting of small doses of sodium nitrite, potassium iodide and intermittent occlusion of the common carotid for fifteen minutes thrice daily, associated with hot compresses and pressure bandaging, the exophthalmos gradually diminished in size and the pulsation was less marked. There was gradually increased motion first inward then upward; several months later, downward and outward. There was developed at the inner upper part of the right orbit, a large vessel, pulsating synchronously with the radial pulse. The left eye did not pulsate and has gradually gone back to its normal position.

The optic nerve in the left side became more atrophic with diminishing calibre to its vessels. The vision remains 5/18.

The condition here had evidently been at first an aneurysmal varix affecting the internal carotid and cavernous sinuses, causing the enormous bruit, which was heard by the patient through bone conduction, causing her great discomfort and transmitted to the auditor with noise of an aortic aneurysm. Its loudest point was just anterior to the junction of the parietal and frontal bone, on a line with the frontal sinuses. The bruit was practically abolished by compression of the common carotid, recurring immediately upon its removal. Much benefit was apparently derived from occlusion of the common carotid for periods of five to fifteen minutes three to four times daily.

After the condition had improved greatly a second and apparently independent varix occurred in the orbit, with the production of a unilateral pulsating exophthalmos, the eye being only temporarily affected.

The pulsating vessel in the orbit is apparently an enormous dilatation of a branch of the ophthalmic artery or possibly as a result of an aneurysmal varix. In view of the good result following ligation of the ophthalmic vein shown in ophthalmic literature, ligation of this vessel may be considered essential to the final cure of the condition. The patient's blood-pressure has gradually fallen from 202 systolic and 125 diastolic, to 168 and 110.

The ocular palsies are disappearing with the return of more normal conditions in the orbit.

Pulsating exophthalmos is a comparatively rare disease, though about 300 cases are recorded. It is frequently the result of an injury.

The lesion may be intra- or extra-orbital. The intra-orbital causes are a true aneurysm of the ophthalmic artery, arteriovenous communication in the orbit, and vascular tumor. The extra-orbital lesions are most apt to be aneurysm of the internal carotid, or perhaps of ophthalmic artery or arteriovenous aneurysm occurring between the internal carotid and the cavernous sinus.

The latter has been found to be the most frequent cause. In an analysis of 19 autopsies, Frost found orbital aneurysm in 3, affection of the cavernous sinus in 2, aneurysm of the intra-orbital portion of the ophthalmic artery in 2, arterior venous communication in 8, and an undetermined condition in 4 cases. I am inclined to the opinion that we had in our case the anteriovenous aneurysm between the internal carotid and cavernous sinus, which explains the symptoms to the best advantage. Later, there was perhaps a new formation of arteriovenous aneurysm in the orbit.

# RETINITIS CIRCINATA

By HAROLD G. GOLDBERG, M.D.
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Retinitis circinata was first described by Fuchs in 1893. He reported a series of twelve cases with symptoms sufficiently uniform in character to be designated an independent affection. According to Oatman ("The Diagnostics of the Fundus Oculi"), the condition was not unknown previous to Fuchs' paper, but was described by Hutchinson, in 1876, as "a symmetrical, central, chorioretinal disease, occurring in senile persons." Subsequently a number of cases were reported as "Hutchinson's changes" and under other names. Oatman claims that the prominent feature from which retinitis circinata derives its name is a girdle of white spots encircling the macula. The characteristic changes usually are confined within the triangular space enclosed between the superior and inferior temporal vessels. In typical cases the macula around the fovea presents a grayish opacity. At a certain distance this opacity is surrounded by a ring of large and small, milk-white spots. Between the ring and macular opacity the retina may or may not appear normal. Generally, the girdle of spots is horizontally elliptical in outline and incomplete on the nasal or temporal sides. The periphery of the girdle usually falls short of the larger temporal vessels and the disk, but in certain cases both may be included within its radius. The vessels pass over the spots when the two are in contact. The spots are not bordered by pigment. When first formed they are round, but may coalesce and produce lobulated forms or even a white band with scalloped edges. The spots give the observer the impression that they are slightly elevated. The diameter of the macular opacity always is greater than that of the optic disk, and in fresh cases it may appear sharply defined. Later, the opaque area may be extended and swelling of the retina is demonstrable. Several cases have been reported in which the retina was detached in the macular region. Pigmentation does not belong to the ophthalmoscopic picture, but stippling and pigment spots have been observed outside the girdle. Within the girdle a delicate haze hangs over the zone of apparently normal retina. In old cases, hemorrhages occur in the affected region, usually along the vessels. According to Fuchs, bleeding occurs in about half of the cases. Holmes Spicer found hemorrhages in the fellow-eye. Fresh spots and new hemorrhages may form. In very old cases pigmentation may occur. The optic disk is unaltered unless touched by the girdle, in which case its outline may be blurred. The retinal veins are tortuous and the vessels may present other arteriosclerotic changes. In course of time the retina within the girdle atrophies. The spots may long remain unchanged or may break up. Fuchs has seen the macular opacity and spots disappear in four years, and in another case continue for seven years. De Wecker has observed a case for twenty years. The spots sometimes change color and exhibit a dirty gray stippling, and cholesterine deposits appear. In some cases a proliferative process ensues and the retina becomes greatly thickened from formation of connective tissue. Oatman continues, that retinitis circinata is a rare disease. Among 70,000 eye patients, Fuchs found 8 typical and 4 atypical cases. De Wecker saw 15 cases among 150,000 patients. In Fuchs's 12 cases only one eye was affected in 7 and both eyes in 5. It appears to be more common amoung women than among men. Generally the subjects are over sixty years of age and have arteriosclerosis; cases occur, however, at all ages. De Wecker saw a case in an infant. Great visual disturbance always exists, but develops gradually. It consists of a relative scotoma, with a diameter of 10 to 12 degrees, which soon becomes absolute. Peripheral vision is not affected, but old cases develop concentric contraction of the field. The light sense remains good. The patients do not complain of night blindness, day blindness, or metamorphopsia, from which it is inferred that the rod and cone layer of the retina is unaffected. The course of the disease is chronic and progressive and the prognosis is bad; but absolute blindness does not result, peripheral vision being retained. The nature of the process and morphology of the spots have not been determined. According to Parsons a variety of opinions have been expressed as to the nature of the spots. Fuchs considered that they consisted of coagulated albuminous exudate, similar to that found in albuminuric retinitis. He opposed the theories that they were caused by arteriosclerosis, and that they were degenerated blood clots. Goldzieher thought the condition caused by arteriosclerosis, the spots were necrotic, due to blocking of ophthalmoscopically invisible arterioles. De Wecker considered that the spots were due to fatty degeneration of extravasated blood, in opposition to Goldzieher, who wrongly stated that the hemorrhages were always secondary. Nuel considered arteriosclerosis the cause, and Henle's layer accountable for the peculiar distribution, while he denied their origin in blood clots. The subject was discussed at the Heidelberg Ophthalmological Society in 1896. Siegrist thought the spots were deposits formed from edematous fluid, and called the disease *œdema retinæ externum centrale.* Only one case has been submitted to anatomical examination, by Ammann. He found hyaline masses in the internuclear layer; some of these showed very distinctly that they were derived from red corpuscles, all the different stages being demonstrable. In the crenations of the nodules there were large mononuclear bladder-like cells, filled with fat globules. Ammann thought it probable that these cells were derived from endothelium of the perivascular lymph spaces, and that they had the function of absorbing the hyaline nodules. The retina was thickened to about four times the normal in the situation of the white spots, the broadening being chiefly in the internuclear layer. The outer nuclear layer and the rods and cones were absent here, but commenced again at the margins of the spots. Müller's fibers were thickened, forming conical expansions in the nerve-fiber layer, the bases of the cones being directed toward the vitreous. The choroid contained thickened and actually sclerosed vessels, the thickening involving the media principally. It must be stated that the eye was glaucomatous. Parsons states that the evidence is insufficient to be positive that all the changes are dependent upon previous hemorrhage. It is probable that albuminous coagula play an important part, for the resemblance to the white spots of albuminuric retinitis cannot be entirely fortuitous, and this is essentially due to edema and not to hemorrhage. It will, therefore, be wise to suspend judgment until other cases have been exhaustively investigated. It must be noted, however, that the condition is not a true retinitis, but is essentially degenerative.

The case I have to report is a male, aged twenty-four years, a Canadian by birth. His father is living and in good health. His mother died of pulmonary tuberculosis. He has had three brothers and three sisters. His brothers are all living and in good health. One brother aged twenty-two has poor sight, for which he has consulted several ophthalmologists, but has never obtained a satisfactory report as to the nature of his difficulty. He has one sister living, aged thirty years, whose only serious illness was diphtheria. She has had eye trouble all her life. While she was teaching school a few years ago her sight suddenly grew worse until she was no longer able to do her work. She was examined by an ophthalmologist and told that she had retinitis. She has improved somewhat under treatment. One sister died one week after birth from an unknown cause, and the parents claim that this child was never able to open its eyes from birth. The other sister died at eighteen years of pulmonary tuberculosis. The case under observation has had in childhood: measles, mumps and whooping cough, and ten years ago a light attack of pneumonia.

He has never had any serious injury, and denies venereal infection. He has always had trouble with his eyes, that is to say his sight has never been keen, but they were never given very much attention until he was eighteen years of age. At this time he was refracted, but according to his statement the glasses never suited him, and his vision was always unsatisfactory. All his life he has been color-blind for green, but has never noticed any night blindness. He is intelligent and well educated, and has made use of his eyes a great deal, at college, and later as a newspaper reporter. He first noticed his more serious eye affection about seven months ago, and he believes it reached its height in about one week. At this time he consulted several Canadian Ophthalmologists from whom he was unable to obtain any satisfactory advice as to his condition, only one of them making anything like a definite diagnosis, and this was to the effect that he would never have good sight. Since then he has been traveling abroad, hoping that the rest and diversion would help him. The patient was first seen by the writer on April 21, 1915. He was apparently in good general health, well nourished; his heart, lungs and bloodvessels were normal; his ears, nose and throat were negative, pulse and temperature normal, urine negative and blood-pressure normal. His blood and spinal fluid were taken for the Wassermann reaction with the following result: blood negative; spinal fluid, 0.5 negative, 1.0 plus 1, 1.5 plus 3. His blood count showed leukocytes, 7360. The study of his eyes revealed the following: right eye, the external appearances were normal; the refracting media clear; in the fundus, the macular region was occupied by a purplish-red area, faintly stippled with darker red streaks; this was oblong in shape, axis 75°, about one-third disk diameter in size. Directly in the centre of this area was seen a glistening yellow spot, pin-point in size, somewhat resembling a fat granule, while immediately surrounding it was a zone of healthy retina, about 3 mm. in breadth. This zone was followed by a well-defined circle of characteristic pale pink to bright white spots, which varied in size from one pin-head to two pinheads in diameter, and formed only one irregular circle completely surrounding the healthy retinal zone. The spots were quite well defined, formed an uninterrupted circle, and showed no traces of pigmentation, either at the centres or at the borders. The remaining fundus was healthy in appearance, the vessels were clean and well defined, and no hemorrhages were present in any part of the field. In the left eye the appearances were much the same as those just described; the purplish-red area was about the same size, it was somewhat more circular in outline, and was not stippled. The glistening yellow point was more pronounced, the intervening zone of healthy retina was broader, and the circle of spots was more clearly defined. These spots were of about the same size, color and form, but existed in greater numbers, and produced several layers, which completely and uninterruptedly encircled the healthy retinal zone. The remaining retina was healthy. The field of vision was characteristic; there was no contraction, but an absolute central scotoma. The patient wore a low, compound myopic correction which had very little effect upon his visual acuity. He was admitted to the Episcopal Hospital and placed under treatment. This consisted of a liberal administration of the mercurials and iodides, and diaphoresis. After a month, some few changes were noted. The purplish-red spots became more decidedly reddish in color, and the white spots less sharp in outline. The glistening point in the macula of the right eye disappeared entirely, while in the left it remained the same. Very little change in the vision was noted; the patient claimed that the letters were more sharply defined, and maintained that his sight had improved, although the visual test did not substantiate this. He was obliged to leave the city and return to his home, so that a further study was impossible.

Six months later the patient returned to this city for observation. There were no changes in the external appearance of either eye, but in the fundus the changes were very decided. The girdle of spots had retained their same general form and arrangement, but they were much lighter in shade. The lines of separation between the zones were much less distinct, the purplish-red areas had faded to a dull red, faintly tinged with purple, the glistening point at the fovea of the right eye had returned. There was a very marked concentric contraction of both fields, an average of fifteen or twenty degrees. In spite of this latter change, however, the condition should be considered improved, the fundus had a healthier appearance, and the vision, which was 2/22 in the right eye, and 2/35 in the left when he was first seen, equals 5/150 in the right, and 4/150 in the left at the present time. With his correction, which is a low compound minus in the right and a mixed astigmatism in the left, his vision equals 5/22 and 5/35 respectively. He was seen by Dr. Francis Sinkler who found that his blood-pressure equalled 150; his urine showed: sp. gr., 1008; albumin, faint trace; sugar, negative; no casts. His spine and vertebræ were negative.

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# FOUR CASES OF OPTIC ATROPHY

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## (1) A Case of Optic Atrophy (with Bitemporal Hemianopsia) in Pituitary Disease

Optic atrophy incidental to disease or faulty development of the pituitary body is well illustrated by the case of J. M. B., (new record system), January 23, 1915. This patient was referred to us by Dr. H. G. Godfrey, of the medical clinic, with the following history:

The patient, a single man, stated that he was twenty-six years of age but appeared to be much undersized and more youthful than that age. About three months previously he had been under treatment in the hospital for subacute appendicitis, for which he declined operation, at which time it was noted that he conformed perfectly to the typus feminis of Froelich, now demonstrated by Cushing to be symptomatic of pituitary deficiency. Dr. Hooker, also of the medical service of the hospital, suggested pituitary tumor. In the early part of the current year (1915) he returned for further observation, largely, however, on account of failing vision.

His family history was negative and his past medical history revealed only acute rheumatic fever in early life, and a successful hernia operation in 1908 in this hospital.

The medical history records (5509 medical dispensary, Dr. Godfrey, new system of records) that he has had severe headaches, accompanied by vomiting, that have lasted at times for five days, and which have occurred at irregular intervals during the past three years. He first wore glasses three years ago. His sexual functions, which have never been active, have been in complete abeyance since his nineteenth year. For the past year he has been somnolent and listless. He has always been very tractable, and his mother thinks he is mentally deficient.

The past medical history as obtained in the eye service brings out the same facts, together with a few sociological details that may have some bearing on the condition.

The patient's first occupation was in a plating mill where nickel and bronze plating by the electro-process was extensively used. This was in Canada, where he worked for five years and two months. He was fourteen when he began the work, and over nineteen when he left it. This work required contact with potash and with cyanide—no wood alcohol, gasolene, turpentine, or benzin was used as far as he knows in the work. He did come in contact with lacquer, which was extensively used in the work. For the last four years he has been working in a carpet mill. Here it may be casually mentioned that the textile industries serve to render many latent optic nerve conditions manifest, as is readily shown by a previous survey of a series of optic atrophy cases.

He had inflammatory rheumatism when three years of age, but never had diphtheria, scarlet fever, venereal disease, or, in fact, any condition other than occasional indigestion, and the hernia previously mentioned for which he was operated upon in the Episcopal Hospital six years ago with a good result.

The patient is unmarried and looks like a lad of less than fourteen years of age. He commenced to have trouble with his eyes about three years ago, for which he attended St. Christopher's Hospital, and for which glasses were prescribed. About one year ago his sight began to fail rapidly, but he did not seek medical attention until within a few days of the first visit recorded in our notes. He has had pains in the head, "bilious attacks," stomach trouble, etc., from time to time, for which he consulted a physician. Divergence has been noticed only the last two months.

The physical examination showed the facies to be curiously bland and unlined. There is left divergent squint. Speech is slow, and response noticeably retarded. The hips are broad. The superficial fat is distributed over the pectoral, abdominal, and gluteal regions so as to give the body a typical feminine outline. The pubic hair is scanty. The chest, arms, and axillæ are hairless. The heart and lungs are negative. The systolic pressure is 110 mm., the diastolic 70 mm.

The laboratory tests showed hemoglobin 82 per cent., erythrocytes 5,170,000, leukocytes 8400 (differential count: polymorphonuclear 58 per cent., transitional ⅔ per cent., lymphocytes 41⅓ per cent.). Blood serum, Wassermann negative. Spinal fluid Wassermann negative—0.5—1.0—1.5 c.c., used. Urinalysis: amber color, acid reaction, specific gravity 1018, no albumin, no sugar, no casts, no crystals.

A skiagraph by Dr. Stewart, radiographer to the Hospital, showed destruction of the sella turcica, probably as the result of a tumor (index number 3997A January 27, 1915).

The first ophthalmological examination showed the eyelids, lacrimal apparatus, conjunctiva, tension, sclera, cornea, iris, anterior chamber, vitreous, and lens to be normal in both eyes. Graefe's sign, Stelwag's sign, Mobius's sign, ptosis, exophthalmos, and enophthalmos were absent.

The pupils were 3 mm. in diameter, round, symmetrical, and equal. Both pupils reacted for light. The right pupil reacted promptly and freely for accommodation. The left pupil reacted during adduction of the left eye toward the median line and during accommodation, but on account of deficient vision in the left eye, the coördinate movement known as convergence was never present and the pupillary reaction for convergence could not be determined. Consensual reactions were present in both eyes. Hippus, Argyll-Robertson reaction, orbicularis reaction, cerebral cortex reaction, and paradoxical reactions were absent. The Wernicke hemiopic reaction, as usual, was absent.

The patient's vision in the right eye was 5/6, taken on two different test cards, but in the left eye it was 1/60, but only in the good part of the visual field. Examination of the fields in a dark room with the electric perimeter showed bitemporal hemianopsia. Color perception was good in the right eye but greatly impaired in the left.

The muscle equilibrium was fairly good, despite the divergence of the left eye. There was no nystagmus. The divergence in the left eye was not due to muscle defect but to loss of ability to fix, on account of the pronounced visual disturbance. Adduction, abduction, elevation, depression, rotation, etc., were good for each eye separately, but there was no convergence of the left eye when accommodating for close range with both eyes.

Examination with the ophthalmoscope showed pronounced optic atrophy in the left eye with atrophic changes in the right eye. There were no pronounced macular changes but the retina was thinned out in the periphery. The choroidal vessels and pigment were also very prominent in the periphery of the fundus. The sclera was nowhere exposed. All the vessels were somewhat contracted, and the main arteries were rather tortuous in both eyes.

The patient was subsequently operated upon by Dr. Charles H. Frazier, at the University Hospital, Philadelphia, February 20, 1915, by the trans-sphenoidal route, and the immediate recovery from the operation was very good.

After allowing sufficient interval to elapse to note any complications, the patient was again subjected to a detailed examination on May 8, 1915. In general he felt much better than before the operation and thought he could see much better also. The developmental changes noted before were uninfluenced by the operation. Nothing was noted externally in connection with the eyes except the divergent squint of the left eye and the slight assymmetry of the face, both of which were present before the operation. The patient at this time was in very good physical condition and expressed a desire to resume work, having elected, during the period of his convalescence, to become a barber.

Examination of the fundus of the right eye showed the nerve to be white and atrophic, especially to the temporal side, but there was no pronounced cupping, although there was beginning choroidal disturbance in the periphery. The left eye showed pronounced optic atrophy but no cupping. The fundus appeared very much hazed, but there seemed to be no other gross lesion.

The pupils were 3 mm., in diameter, equal, symmetrical. The reaction to light was good in the right eye but retarded in the left. The right reacted to accommodation, convergence, and consensually. The left reacted to accommodation and consensually, but not to convergence, since there was no convergence of the left eye.

The gross muscle movements were normal but there was no convergence of both eyes simultaneously. That this may occur in other conditions has been noted by Roemer (*Text-book of Opthalmology*, iii, 607) and others, the observation being that the tract for innervation of convergence may be affected independently of that which transmits the innervation for the lateral movements. In this case we believe the cause was peripheral rather than central, and depended primarily on the loss of vision incidental to the optic atrophy. Otherwise we would have to assume the presence of a lesion in the crura cerebelli, in the pons, in the corpora quadrigemina, or in the optic thalamus, and we have no other evidence to support this speculation.

The vision of the right eye at the time of this examination was

5/5, and at close range of 35 cm. he was able to read the 0.50 m. type. The left eye was able to identify the largest letter on the chart, marked for 45M, at one-half meter distance. The fields showed distinctive changes, which are well expressed by the charts made on this date.

Since these last notes were made the patient has developed some new symptoms that point to a return of the intracranial trouble and has been referred to Dr. Godfrey in the medical clinic, who has instituted treatment with some of the pituitary derivatives. The case is still under his observation.

## (2) A Case of Retinitis Pigmentosa with Optic Atrophy

The patient in this instance was a man, aged about thirty-two years, whose chief complaint was his inability to see even the larger objects as night approached.

The medical examination by Drs. Robertson and Belk, while the patient was in the hospital, was especially comprehensive, and served to eliminate many factors of possible etiological significance. The history showed that the patient's mother was living and well, and that there were no evidences, insofar as history could show, of maternal specific disease. The father had died some time since of pneumonia. One sister was living and well, and constituted the sole support of the family. The patient did not recall having had any of the usual diseases of childhood, and his general health had always been good. He was born in England but had been in this country for the past five and a half years. He had used tobacco sparingly, but had totally abstained from it for the last two years. He denied the use of alcohol.

About three years previous to his coming under our observation he noticed a failing of his sight especially toward evening, which was manifested by his bumping into gross objects. This was accompanied by pain over the brows and by nervousness, so pronounced that he was compelled to give up his work. Since that time, however, he has had no pain. This loss of vision had become gradually but progressively worse, so that now he cannot see to go about by himself.

On admission, the examination showed the ears, nose, throat, and mouth to be apparently normal. The heart and lungs were also found to be negative. The abdomen showed nothing abnormal, and the extremities were characterized by an absence of disease or deformity. All the reflexes were found to be increased, especially the biceps and patellar. Ankle-clonus and Babinski were present. The gait was spastic.

Dr. Robertson's examination showed nothing abnormal as regards the heart, lungs, or abdomen. All the reflexes were increased. Regarding the mental condition, he expressed himself of the opinion that, during childhood, the patient had probably had an acute infection, such as scarlet fever that caused a low-grade pachymeningitis, which did not develop enough to give definite manifestations, but simply sufficient to cause the dull and impaired mentality in this case.

Examination of the ears, nose, throat, and sinuses by Dr. Biedert was entirely negative. A sclerotic rhinitis was present but there was no sinus disease.

The laboratory data afforded by this case read:

Blood Wassermann, negative. Blood picture: hemoglobin, 91 per cent.; red blood cells, 4,790,000; white blood cells: leukocytes, 12,720; polymorphonuclears, 56 per cent.; mononuclears, 0.3 per cent.; transitionals, 2.0 per cent.; lymphocytes, 41.0 per cent.; eosinophiles, 0.6 per cent.;

Urinalysis: Amber color; acid reaction; 1020 specific gravity; flocculent; trace of albumin, faint ring by contact tests; sugar absent; casts, occasional hyaline; crystals, amorphous urates; leukocytes, negative; erythrocytes, negative; bacteria, many; epithelium, many.

Spinal canal fluid: Wassermann, negative; Nonne, negative; Noguchi, negative; Fehling's reduced; cell count 3 to 1 cm.

The history and data obtained from the ophthalmological examination are also of interest. The patient was born in England of very poor parents, and was not able to walk until two and a half years of age, and was not able to talk plainly until fifteen years of age. His dialect is that of the working class of Lancashire, England, where he was born, with the additional peculiarity that he pronounces letters like "s" as though they were "t." There is no history of convulsions in childhood. There is no consanguinity of parentage.

The patient never had a skilled trade but worked as a general utility man in whatever factory he could get work. His mother states that he never smoked or drank, and had always led a good "Christian" life. When he was nineteen years old he had glasses (—S. 7.00 both eyes) prescribed for him by a Dr. Walker, in Liverpool. These he has since continued to wear, with comfort, until about three years ago. It was then that he noted a marked impairment of vision which compelled him to stop work. This failure of vision was more noticeable at the close of the day's work, and he was continually bumping into things on his way home from work. Coincident with this he had pains in the head, and subsequently, he was quite morose and melancholy. His station and gait were, and now are, still good. About the same time as the failure of sight occurred, it was noticed that he became more or less deaf, or at least slow of comprehension. This is still present, and while there is no deafness present now, words and statements frequently need to be repeated once or twice before he grasps their meaning. His replies are always logical, and he shows no pronounced mental deterioration. He appears to be "poisoned" more than anything else. His teeth are poorly developed and rachitic, and there is a pronounced pyorrhea.

The detailed ophthalmological examination revealed several points of interest. The vision in each eye was the perception of fingers at eight inches. By retinoscopy, it was determined that myopia to the extent of nine diopters existed in each eye. With this correction, his vision was brought to 5/15 in each eye. The fields were obviously contracted, as was readily demonstrated by gross tests, but perimetric examinations were impossible on account of the myopia, and the dull mentality of the patient. Central color perception did not seem to be impaired.

The eyelids, lacrimal apparatus, conjunctiva, sclera, cornea, and iris appeared to be healthy in each eye. The tension was also normal in each eye. The anterior chamber was of normal average depth. The aqueous, lens and vitreous were transparent.

Graefe's sign, Stelwag's sign, and Mobius's sign were absent. Ptosis was not present, and there was no exophthalmos or enophthalmos.

The gross muscle-balance appeared to be perfect, at least there were no palsies or gross defects. Nystagmus was absent in each eye.

The pupils were equal and contracted and inactive. The unusual reactions, as well as the usual ones were not to be demonstrated.

The examination of the fundus of both eyes showed practically the same condition in each, namely pigmentary degeneration of the retina with optic atrophy. The nerves were in the process of simple atrophy and did not show the changes that accompany high myopia. The coats of the eye were greatly stretched, as in high myopia, the choroidal circulation being especially prominent.

The retina showed the stellate pigment islands characteristic of the pigmentary degeneration of that structure. The area occupied by the macula seemed to be free from disease.

The case illustrates no rare feature of the disease except the elimination of the usually accredited etiological factors by scientific investigation. Consanguinity of parentage was readily eliminated, but it remained for the careful laboratory work in this instance to remove from the etiology any possibility of specific disease. This is probably the first American case to be handled so thoroughly.

Various attempts have been made to improve these cases by treatment along the lines of special medicines and other agents, but little is ever accomplished by reason of the fact that the patient's social and economic condition permits of so little change in his occupation or environment. In this case, the patient was permitted to give up his work, through the self-sacrifice of his sister, and, by remaining quietly at home, doubtless checked the advance of the degenerative process. Careful objective refraction also served to accurately determine his error of refraction and in this way gave him more vision and thus relieved the eye of the congestion incident to eyestrain. With the assistance of the Society for Organizing Charity and its allied agencies, we are trying to find special work for this man, work that will be practicable for him to pursue in view of his infirmity. This work will also serve as a therapeutic agent, if obtained, by relieving him of his melancholy tendencies.

### (3) A Case of Optic Atrophy with Exophthalmos Incidental to Benign Intra-orbital or Intracranial Neoplasm

A pronounced case of exophthalmos with optic atrophy, which was assumed to be due to some benign intraorbital or intracranial growth, was observed in the person of S. B., (vol. ii, p. 643; November 1, 1913). This case also presented other interesting features not included in the above title, and it is for this reason that it is thought worth while to place the notes on record:

The patient was a man, forty-three years of age, and a designer by occupation. He presented himself to us by reason of a pronounced swelling of the left eye. Examination revealed that the eye was not in itself swollen, but that it was protruded down and somewhat out by reason of swelling and infiltration of the orbital tissues. The right eye, also, was very slightly proptosed. The condition in the left eye had extended over a period of three years, at times not so pronounced, but had given him no concern until vision had begun to fail in the right eye, which was about one year previous to his coming to this hospital.

In looking over our previous records in order to verify the patient's statement as to his chief complaint we find that he had applied to the clinic within the period mentioned, but only for the relief for beginning presbyopia, and the symptoms now present were not in evidence at that time.

The vision at his first visit (for the condition now under discussion) was: right eye without correction, 5/4.5; with correction, 5/6; left eye without correction, 5/200; with correction, 5/200. His chief complaint in addition to the "swelling" of the left eye was blurring of vision, and this blurring of vision extended over a period of nine months, greatly interfering with his work. Our detailed examination at this time showed the eyelids, lacrimal apparatus, tension, conjunctiva, sclera, cornea, iris, anterior chamber, lens, and vitreous to be normal. There was a slight bulbar conjunctival congestion. The iris was of a bluish-gray color. Graefe's and Mobius's signs were absent. Stelwag's sign and exophthalmos were demonstrated in the left eye. Ptosis was absent. The pupils reacted to all stimuli but the left was comparatively inactive. The Wernicke reaction was doubtfully present at times in the left eye. Otherwise the pupils presented no departures from normal. The gross muscle balance was normal and no pronounced defects were demonstrable.

Inquiry into the patient's personal history elicited the usual amount of useless information, from which we determined that he drank an occasional glass of beer, and that he never had any venereal disease. Muscular rheumatism was his sole illness prior to the present condition. One of his earliest symptoms was deafness and "ringing" in the right ear, which had been continuous for the last two years. He wore glasses in order to relieve the weakness, as he expressed it, of the left eye, but without benefit. This trouble had been ushered in three years previously by an attack of vertigo. There had been no previous or subsequent other trouble in walking or coördination, but he was unable to find the small squares in the designing paper on account of failing vision, failing color-sense, and sense of proportion. There were no motor troubles and no diplopia. There was occasional photophobia. Nausea was present with the vertiginous attacks, which had numbered as high as twenty or thirty in a week. The temporal arteries were very much dilated, especially the right temporal artery.

Detailed examination of the muscle balance at this time showed 9 degrees interni; 9 degrees externi; ½ degree inferior recti; ½ degree superior recti. Maddox rod showed 1 degree exophoria; ½ degree hyperphoria; prism base up right eye. No diplopia with candle test in the dark room.

The fields showed some interesting phenomena. The right eye showed normal field for white with a relative scotoma to the temporal side on the 120 meridian between 50 and 60 degrees. The red field was contracted to 15 degrees; the green within 10 degrees. The left eye showed a normal field for white with disseminated scotomata above fixation in segment 15 to 18 degrees. Green was irregularly contracted between 20 and 10 degrees. Red was irregularly contracted within 10 degrees with central scotoma.

The fundus showed atrophic changes in the nerve, accompanied by pronounced enlargement of the retinal vessels especially on the left side. At the first visit (November 1, 1913) the ophthalmoscopic examination showed the following conditions. In the right eye the optic disk was oval, of a pinkish-white color, and well defined. There was a deep excavation to the outer side. Vessels long axis minus 1.00 D. The arteries were normal in width, rather numerous; the light streak was normal; and numerous arterioles were visible in the fundus, as the periphery was approached. The main veins were rather distended. There were no hemorrhages or fatty deposits.

In the left eye the optic disk was rather oval at 90 degrees, with its superior and nasal margins somewhat obscured, which obscuration could not be cleared away with the lenses of the opthalmoscope. A central physiological cup was discernible. There was present enormous distention and tortuosity and kinking of all the retinal veins, while the arteries appeared to be abnormally contracted. A small hemorrhage was present along the course of the inferior temporal vein. There was pronounced choroidal pigment to the temporal side of the disk. In both eyes the macula was normal. No edema of the retina was present in either eye.

Examination by Dr. C. C. Eves in the nose and throat clinic showed a small frontal sinus on the right side, left sinus normal, no cloudiness. Transillumination was entirely normal.

The *x*-ray examination by Dr. Stewart confirmed transillumination, and further showed the sella turcica to be normal though small.

The laboratory examinations showed Wassermann test negative and a blood count of leukocytes, 10,720; polymorphonuclears, $77\frac{1}{3}$; transitionals, $3\frac{1}{3}$; lymphocytes, 18; eosinophiles, $1\frac{1}{3}$.

Twelve days after his first visit the left eye showed normal reaction to light, with normal consensual reaction on both sides. The ophthalmoscope at this time showed the retinal veins of the right eye to be enlarging.

Examination of the hearing showed the watch to be heard two inches from the right ear and twenty-four inches from the left ear. The patient, however, complained of faulty hearing in the left ear and "ringing" in the right ear. He also showed a tendency to fall to the left when the vertiginous attacks came on. His temperature remained normal throughout the course of the condition.

Investigation in the medical clinic by Dr. Godfrey disclosed the fact that the patient had been treated there two years previously for cardiac hypertrophy and vascular hypertension. Examination at this later visit revealed the presence of chronic interstitial nephritis and cardiovascular disease. His systolic pressure was 164; diastolic, 100. There was cardiac hypertrophy and relative incompetence of the mitral valve. There were no sensory disturbances. Knee-jerks were both ++, and ankle-clonus was present on both sides.

Despite the fact that the Wassermann test was negative, and on account of the increasing severity of the symptoms, empiricism was permitted to enter into the treatment of the case, and the patient was placed upon potassium iodide, grains 5, three times daily, gradually increasing the dose.

Within two weeks the nausea, vomiting, headache, dizziness, and vertigo were pronouncedly influenced. The dizziness disappeared entirely. Within the next two weeks, all these subjective symptoms had disappeared. The exophthalmos and proptosis were less marked, but the distention of the bloodvessels was still present. Symptoms of iodism demanded a reduction in the dose of the potassium iodide.

Five weeks after his initial visit our notes read that he had had an attack of dizziness the last week (corresponding to the period when the amount of potassium iodide had been reduced). Ophthalmoscopic examination showed in the right eye that the media were clear, the disk was oval at 90 degrees, scleral ring was all around, choroidal ring in and out, broken to the outer side; the veins were very full and more tortuous than normal and twice the caliber of the arteries. Physiological excavation of the disk was present. There were no hemorrhages or exudate. In the left eye the media were clear; the disk was irregular; the excavation was pronounced, small and tortuous vessels were present on the disk. Dark and tortuous veins, much larger than in the right eye, were seen emerging from the center of the disk; the arteries were contracted and were less than one-fourth the diameter of the veins, especially in the lower branches. In the periphery they were somewhat larger.

Potassium iodide was administered continuously, first in increasing doses until symptoms of iodism required decreasing it, then, for the most part in doses varying from 5 to 15 grains, three times daily in water, after meals. Two months after our first observation his vision was right eye, 5/45; left eye 5/200. Vertigo and dizziness had practically disappeared and the proptosis and exophthalmos were markedly reduced. The left eye showed a marked diminution in the venous engorgement, although the arterial twigs were still quite large. There was a minute hemorrhage along the superior temporal vein anterior to the macula.

At the end of four months from the initial visit the right eye had still maintained its normal visual acuity and the left eye had increased to 5/60. Hearing had increased to watch sounds before the left ear at two feet. His weight also increased. The patient had returned to his work, the vertigo and dizziness having been reduced to negligible proportions. The proptosis had nearly all disappeared. The pupils reacted freely to all forms of stimuli. The venous engorgement, so prominent in the other examinations, while still present in the fundus, was considerably less marked than formerly.

On looking further into our records we discovered that the patient had been under our observation in October, 1911, two years previously (vol. 1, No. 245). He then complained of dizziness, and he was refracted. The ophthalmoscope showed in the right eye no gross lesion but tortuosity of the vessels. In the left eye the superior nasal vein was quite tortuous. The other vessels were also tortuous but less marked. The veins were slightly irregular in course and depressed where crossed by the arteries. There was no gross lesion in either eye. Proptosis was absent.

Sixteen months after the initial examination for the present condition was made the fields are found to be practically normal for white, red, and green, with the exception of slight interlacing on the nasal side. There are no scotomata. Vision in the right eye was 5/6 and in the left eye 5/22.5, without correction. The exophthalmos and proptosis, so pronounced in the early stages of the condition, have disappeared, and the circulatory disturbances previously existing within the eye have subsided. The patient has continued at his work after his return to it, already noted, and the subjective symptoms of dizziness, etc., are no longer experienced.

## (4) A Case of Simple Optic Atrophy of Specific Origin in a Child

Simple optic atrophy occurring in a child, thirteen years of age (new system of records, card No. 1018, February 2, 1915) as the result of inherited or congenital syphilis was also observed in this clinic. The patient was a schoolgirl and gave a very vague history of a still more vague illness dating back one year (December, 1913), in the course of which she suffered with "nervousness," vertigo, neuralgic pains in the head, etc., causing her to be away from school, and at times to be confined to bed. She also had trouble in walking about, due to giddiness and vertigo. Previous to this attack her vision had been normal. In no way did the illness resemble meningitis of any variety, but during the course of it she had difficulty in seeing out of the right eye, which difficulty disappeared after her recovery (about March 1, 1914). When she returned to school, she noticed failing vision in the left eye, which became progressively worse, although she was under observation and treatment at this period at St. Christopher's Hospital, Philadelphia. The record here (Book No. 24, p. 6) shows that in May, 1912, her vision was 5/8 and 5/10, and that she had been sent from school by the school inspector on account of this deficiency in vision. Glasses were prescribed without benefit. Her attendance at the latter hospital was somewhat irregular, and she was not under continuous treatment until recently.

In the latter part of 1914 it was noticed that the patient showed evidences of atrophic changes in both nerves, but especially pronounced in the left eye, the vision of which rapidly dropped to zero, with intermittent light perception.

The record of the detailed examination on December 2, 1914, shows:

Right eye: Vision 5/6; lids and conjunctiva normal; cornea clear; pupil in repose semidilated; reaction to light, accommodation, and convergence normal, prompt and free. No gross muscle palsies, although when both eyes are used there is a slight paretic movement in the periphery. The media are clear, the disk is round with a broad cupping extending to the temporal border, very deep and atrophic in appearance, although the nasal side of the nerve head is not cupped and is pinkish in color. The choroidal ring extends almost completely around the nerve except at its superior border. The vessels are normal in character and course; they may be said to be somewhat distended if at all altered but in no instance are they contracted in any part of the fundus. There is slight choroidal disturbance in the periphery of the fundus. Otherwise the fundus of this eye is normal.

Left eye: Vision is reduced to the faintest perception of concentrated light in the dark room. There is slight divergence. There are no gross muscle palsies, but when both eyes are employed together there is slight paresis in the periphery of the field of motion. The pupillary reaction to light is almost absent but may be demonstrated by the use of the magnifying glass in the dark room with concentrated light. Consensual reaction for light, accommodation, and convergence is obtained, but when the left eye is used alone there are no pupillary reactions demonstrable except as noted for light. The media are clear; the nerve is round and decidedly atrophic with a broad and deep atrophic excavation; the vessels are distended and tortuous on the disk, especially the central retinal vein, but at no part of their course are the vessels contracted. The choroidal ring is complete but otherwise there is no gross lesion except a slight haziness of the retina.

A later examination (by F. K.) showed in the right eye that the pupillary reactions to light, accommodation and convergence were normal. Ocular movements were slightly paretic in the periphery. There was no gross lesion of the fundus.

In the left eye there was a very feeble reaction to light if any. There was no reaction to light, accommodation or convergence except when both eyes were used together thus showing the reactions to be consensual. The fundus showed atrophy of the optic nerve. The nerve was bluish-white in color; the arteries and veins were full, and this fullness extended to the periphery.

It was also noted at this time that the knee-jerks were slightly plus. The temperature was taken in order to eliminate any infectious process still in existence and was found to be normal.

The hearing was examined likewise, the patient having complained of ear-ache from time to time. Recently she had had an attack that had lasted for three days. At the time of this examination, hearing by air conduction on the affected side (left) was much diminished for the middle C tuning fork. Bone conduction was increased. The high-pitched fork was diminished by about one-half as was also the low-pitched fork. The right side showed no departure from normal. The left ear also showed upon examination a whitish purulent discharge with odor, in the external auditory meatus. This condition had been of indefinite duration. The patient denied ever having had scarlet fever or measles. After the removal of the discharge, the examination showed the drum bulging in the upper and posterior portion; the handle of the malleus was obliterated by the inflammation. Immediate incision was advised and performed. The right ear was normal. This condition served only to add some confusing data in the study of the case, and was subsequently shown to have little direct relation to the real condition under consideration.

The examination of the nose showed the right side somewhat involved in the process described in detail in connection with the left side. The left side showed purulent discharge beneath the middle turbinate which was considerably enlarged. Pressure beneath both superciliary regions elicited tenderness worse on the left side. Pressure on the sides of the nose at its base also elicited tenderness worse on the left side. This suggested bilateral frontal sinusitis, and ethmoiditis on the left side, but these symptoms were not constant and could not be demonstrated with any degree of regularity. Transillumination showed all the sinuses to be unaltered from the normal.

The tonsils had been removed at some remote period (two years previously at St. Christopher's Hospital). There was present a postrhinopharyngeal discharge. Remnants of adenoids were present, and there was also evidence of a traumatic amputation of the uvula.

The child was admitted to the Episcopal Hospital in the early part of 1915, and subjected to various detailed examinations to determine the cause of the optic atrophy. The nose, throat, and ear examinations by the head of this department seemed to indicate the presence of a low grade of inflammation within the frontal (right side), ethmoid, and mastoid (left side) sinuses, a view not concurred in by us, or demonstrated by the several skiagrams subsequently taken of the patient.

At the time of admission the fields in the right eye were fairly good but six weeks later despite active treatment and increase in central vision they were markedly contracted.

The Wassermann test made at this time was positive +4. Spinal Wassermann was negative.

Inunctions of mercury were instituted immediately, and within a few days the patient was ptyalised. Neosalvarsan was also given, later.

After a period of three months of treatment the Wassermann test was found to be negative. The vision of the right eye increased to 5/5. In the left eye, hand movements and light perception was obtained at times at a distance of six inches, but did not remain constant. The atrophic process seemed to be checked and the nose and ear conditions abated so that they were no longer apparent.

# ATROPHIC RHINITIS, TREATMENT WITH SCARLET RED[1]

By W. C. WOOD, M.D.
ASSISTANT IN THE AURAL AND LARYNGEAL DEPARTMENT OF THE HOSPITAL

In the October, 1913, issue of *The Laryngoscope*, Dr. K. K. Wheelock, in an article entitled "A new and efficient treatment of atrophic rhinitis," cited the results he obtained in a number of cases which he treated with scarlet red. These results so far surpassed any which I had ever been able to obtain by the various methods of treatment and with the numerous applications which have from time to time been suggested, that I decided to try this dye on the atrophic cases which were visiting the dispensary.

We all recognize the fact that the more numerous the drugs recommended in the treatment of any given condition the less do we usually know about that condition. This has been particularly true of atrophic rhinitis, and I believe that most of the benefit which has been derived in the past has been due not so much to the particular drug or preparation used but rather to the thorough cleansing of the parts at regular and frequent intervals. I have failed to produce any marked change in the appearance of the mucous membrane itself and have always seen a return to the previous disgusting condition just as soon as treatment has been discontinued. This has been due, I believe, to the fact that so little has been definitely known as to the etiology of the condition, and as proof of our uncertainty of the etiological factors we have only to go over the various tables as given by the authorities on the subject.[1] Perez[2] claimed to have discovered a specific agent, and asserted that every case originated by infection from another patient or from the muzzle of a dog. De Simoni,[3] on the other hand, has "inoculated the normal mucous membrane with the Loewenberg-Abel bacillus, the diplococcus lanceolatus of Fraenkel, and also with crusts from cases of ozena, and in no instance did he succeed in reproducing the disease."

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[1] Read before the Episcopal Hospital Clinical Society, Philadelphia, March 16, 1914. (Reprint from the Laryngoscope, St. Louis, May, 1914.)

Congenital narrowness of the nasal fossæ, undue patency of the nasal chambers, and arrest of development of the lower turbinals have been given as possible structural causes, but I believe they act only indirectly as factors in the production of atrophic rhinitis. Others claim it to be secondary to purulent rhinitis of childhood, ethmoidal osteitis, rarefying osteitis of the lower turbinals, accessory sinus disease, focal suppuration in the nose, accessory cavities, or postnasal space, atmospheric desiccation and bacterial invasion.

Considering carefully the findings of the many observers, and considering also the clinical evidence presented by the cases which we see ourselves, the whole matter seems to narrow itself down to some condition which produces a mucopurulent discharge in the nasal chambers. If this be true, the following group of conditions would seem to be responsible, directly or indirectly, for the chronic rhinitis in question:—Congenital, developmental, or acquired deformities resulting in a widely open or extremely narrow nasal chamber, suppuration from the accessory sinuses, and growths in the nasopharynx with suppuration.

There is no doubt in my mind that the mucopurulent rhinitis of childhood precedes the atrophy in a great majority of cases, and that this discharge is the result either of a large adenoid or of suppurative sinus disease, or both, seems all the more probable when we remember the following facts:—

"Suppuration in the frontal and maxillary sinuses seldom gives rise to symptoms of ozena, but it is not at all uncommon for pus in the sphenoidal and posterior ethmoidal cells to do so. Now these cells are present at birth and are close to the post-nasal space, so frequently the seat of suppuration in early childhood."[4]

With these facts before us, it would appear to me that the atrophy is not due directly to the deformity or obstruction present, with the resulting limited or increased space in the nasal chamber, but rather to the fact that the mucous membrane is being constantly bathed with a muco-pus, this going on over a long period of time, attended naturally with a chronic inflammatory condition, with circulatory and tissue changes and finally the atrophy, loss of sense of smell, altered secretion, and consequent crust formation.

So much for the etiology of the condition. Now as to the treatment of these cases. Once the condition has been recognized, it becomes our duty immediately to determine, if possible, the underlying cause and to remove it. A deviated septum should be straightened as soon as the chambers have been made reasonably clean; the mucopurulent discharge, if still present, should be traced to its source and that region treated. Should there prove to be a large adenoid in the nasopharynx, it should immediately be removed. If, on the other hand, the discharge comes from one or more of the sinuses, the condition should be properly and immediately treated, either by washing and applications, or by surgical means as the case may demand.

Having either failed to find the original cause of the atrophy, or having found and removed it, we must now turn our attention to the atrophic condition itself, and it is here that we have, as a rule, been unable to effect a cure. The removal of the cause, where it has been found, and careful treatment afterward has in many cases benefited the patient, at times arresting the process, stopping to some extent, at least, the formation of crusts, and decreasing or even eradicating the ozena, yet the atrophy of the tissues themselves remained, and many of the minor, yet disagreeable symptoms, persisted.

It is a peculiar fact that in a large number of cases, the atrophic condition is called to our attention for the first time long after the original cause has disappeared, and in spite of the fact that the patient must have had catarrhal symptoms over a long period of time. However this may be, the large majority of cases of atrophic rhinitis which we see present simply an atrophy, ozena, crust-formation, and in many cases, especially those of long standing, a loss of the sense of smell, and it is this end-group of symptoms which has seemed almost hopeless, requiring treatment over an indefinite period of time, and which relapse to the former disgusting condition just so soon as treatment is discontinued.

I have followed Dr. Wheelock's suggestion and used scarlet red in all the atrophic cases coming to the dispensary; and I believe the results obtained are just as noteworthy as those in his cases. Whether these cases will relapse after the discontinuance of treatment or not remains to be seen, but there is no doubt the results obtained over the short period of time I have used scarlet red far surpass any I have been able to obtain in the past. No case failed to show marked improvement in two weeks after the initial treatment. The crusts almost entirely disappeared in this length of time and continued to decrease in quantity with each treatment, until they finally disappeared. The mucous membrane resumed its normal color and thickness, and the ozena was no longer noticeable. This has been the history of all the cases under treatment and I will outline briefly the history of one of the most marked of the group.

Mrs. J. B., aged forty-six years; injury to nose at age of ten years. This was followed, evidently, by a profuse purulent discharge which lasted about three years. The discharge then decreased, but she says she has had "catarrh" with some discharge ever since. Some three years ago the crusting and ozena became so marked that she applied at one of the dispensaries here in the city for treatment. She first came to the Episcopal Hospital dispensary about one year ago and at that time presented the typical symptoms of atrophic rhinitis. The crusts were large, forming casts of the nasal chambers; there was a marked ozena and an entire loss of the sense of smell. She stated at that time that she had not been able to detect odors for about two years. Treatment was immediately started, the chambers being thoroughly cleansed and various applications made. The application then used, which seemed to do the most good, was a saturated aqueous solution of potassium permanganate, but the crusts and ozena never entirely disappeared, and at no time did she regain the sense of smell.

About December 1, 1913, I started using scarlet red and the results were almost incredible. The crusts and ozena disappeared, the membrane itself thickened and regained its normal color, and to my surprise, after about six week's treatment, she told me she was able to smell. I held a bottle of ichthyol under her nose and, with a look of disgust on her face, she remarked that the odor sickened her. I am, therefore, very naturally delighted with the results so far obtained and only hope that they may be as lasting as they were prompt.

In closing I will give an outline of the technic employed.

In the first place I originally applied the scarlet red ointment which is used here in the surgical dispensary. This I am told is a 10 per cent. ointment. I found, however, that was too stiff, rolling up and falling from the turbinals as fast as it was applied. The druggist then thinned it down for me with liquid petrolatum until we had an ointment which would spread nicely over the turbinals and adhere to them. This resulting ointment was a little over 5 per cent.

I first applied hydrogen peroxide to both nasal chambers and thoroughly removed all crusts. The nose was then carefully sprayed with an alkaline, antiseptic solution, after which the scarlet red was applied to every portion of the mucous membrane in the nose, and the patient instructed not to blow the nose immediately, but to wipe the secretions away as they appeared at the external nares. There seemed to be no noticeable reaction after the application, nor did the patient complain of any disagreeable sensations.

After a few treatments, however, the patient notices a stinging sensation after the scarlet red has been applied, which, together with the return of the sense of smell, would seem to indicate a regeneration of the nerve filaments, an observation made also by Dr. Wheelock in his report.

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# REPORT OF THE DENTAL AND ORAL SURGICAL DEPARTMENT FOR 1914

By JAMES R. CAMERON, D.D.S.
RESIDENT DENTAL SURGEON IN THE HOSPITAL

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This department of the Episcopal Hospital was inaugurated March 1, 1914.

A fully equipped dental operating room is situated in the dispensary building, and each afternoon, from 2 P.M. on, is devoted to the out-patient service of the hospital and the mornings to the treatment of house cases.

Unfortunately, the department as yet has not an oral surgical chief. The appointment of a visiting chief I hope to see brought about before the completion of my term of service as resident dental surgeon.

The graduate in dentistry is not yet as enthusiastic in regard to a hospital appointment as is the medical graduate—therefore in offering inducements for a man to carry on this important work the character of training to be obtained in surgical diseases of the mouth, jaws, and associate parts will be the deciding factor. This can be accomplished only by the appointment to the hospital staff of visiting surgeons—an oral surgeon—who will guide and supervise the work of the dental resident.

The dispensary work is more than one operator can handle, patients having to be constantly turned away. Could the dental operating room be enlarged so as to accommodate three or four operators there would still be a surplus of clinical material.

In this report I have endeavored to present cases of interest to the practitioner of medicine. It will be seen that the department does not confine itself to the dental organs alone but includes also the surrounding parts. The field of modern dental surgery is expanding with marvelous rapidity, and it seems as though the full medical course for the practice of this specialty is looming large in the distance. The work of the dental practitioner should go hand-in-hand with that of the medical man, especially with that of the rhinologist. It is a recognized fact today that the rhinologist looks to the dental surgeon for aid in establishing normal respiration. The dentist practising orthodontia has at his command the power to stimulate bone and widen the dental arches, thereby enlarging the floor of the nose. Meati are enlarged, septal curvatures modified, and the restored function of the organ encourages normal growth and development.

The dental surgeon should recognize his responsibility as the custodian of the oral cavity—the gateway of the body. He should guard against the formation of nasal deformities that will surely occur if malocclusion is allowed to continue, also against the ever-present possibility of the oral cavity becoming pathological to such an extent that systemic disturbances follow. Take, for instance, a patient who is addicted to the habit of mouth-breathing. On examination we invaraibly find enlarged tonsils, adenoids, and narrow contracted arches. The patient is not strong—is dull, sullen, and backward with his work at school. Of what permanent value to that patient is the removal of his tonsils and adenoids *if his dental arches are untouched?* The malocclusion present makes it a physiological impossibility for him to close his mouth; hence, he *remains* a mouth breather.

Pyorrhea alveolaris can be found in a great many chronic arthritis cases, and when the mouth has been returned to a healthy condition the system reacts and the patient with the aid of medicinal assistance soon recovers. Too often gingivitis is called pyorrhea. There is a great difference between the two diseases. Pyorrhea alveolaris may be characterized by the pus-producing, inflammatory condition surrounding the teeth and involving the alveolar process. The chief predisposing causes of this disease may be summed up as deposits on and around the necks of the teeth, malocclusion, faulty operative procedures, and lowered vitality of the patient.

Let us now go over the treatment of *fractures of maxilla and mandible.* From the review of literature on this subject there appears to be no universal method in practice for the treatment of these cases. I feel confident that the method of using interdental and intermaxillary splints is by far the most satisfactory. The Barton bandage is only satisfactory as a means of temporary relief. Unless there is no displacement present, and the articulation good the Barton bandage will fail entirely in its purpose. A fractured jaw that has poor articulation and a bad displacement will not be held in approximation long, if at all, by a bandage.

Wiring by means of silver wire passed through the body of the mandible is not a good procedure to carry out, as it invites infection and rarely ever gives good approximation of the parts and restoration of occlusion. Necrosis and disfigurement is often the result following this form of treatment. The use of Lane's plates has the same objections as has the wiring method. The fact of a scar being present even if necrosis does not take place is sufficient in itself to condemn the method when there 's a better and easier one at hand.

The interlacing of the teeth and fixing of the upper jaw to the lower by means of wire sutures is at times a satisfactory means of treatment providing there are good sound teeth present. The objection to the use of this method is the tendency to produce pyorrhea alveolaris.

The interdental splint is made of either gold or German silver. An impression is taken of the upper and lower jaws in plaster of Paris or modelling compound. The impression of the jaw not injured is taken first; this gives the patient confidence and tends to allay his fears. The impression of the fractured jaw is now taken and both impressions are cast and models obtained. The model of the fractured jaw is cut and reduced to normal occlusion with that of the upper. Dies and counter-dies are made of zinc and lead respectively. The metal decided upon is now swaged into place—two laminas being used and soldered together. We now adjust splints to the teeth of the affected jaw and do any adjusting necessary. With everything correctly placed the splint is cemented to the teeth at the time of reduction. A Barton bandage is worn for twenty-four hours or so and then removed. The splint is to be kept on from five to six weeks, the patient presenting himself for prophylaxis twice a week. By the end of the sixth week the bone will have united firmly and the splint may be removed.

The great advantage of this method is its convenience to the patient, inasmuch as there is no need of wearing a bandage for a lengthy period; and further, the patient is not advertising the fact that he has sustained a fractured jaw.

The service has been extremely fortunate in the number of fracture cases admitted, there being thirty-three in all. Two of the patients died soon after admission to the wards, having received other injuries. With the exception of one or two cases all have been treated by splints with most gratifying results.

In conclusion, I, as first resident dental surgeon to the hospital, wish to thank the visiting surgeons, physicians, superintendent, residents, and the late chief resident physician, Dr. T. G. Aller, for their helpful advice, and courtesies extended, always willingly given for the establishment of this department on a firm basis; and the opportunities afforded to its resident for the study and following up of cases in the general medical and surgical wards.

## REPORT OF CASES.

CASE I.—*Sepsis following alveolar abscess, unrelieved by operation.* (Service of Dr. H. C. Deaver.)

The patient, a married woman, aged forty-two years, had always been healthy, and had raised a large family. She first complained of toothache in the lower left second molar. The following day her face became swollen, and she sought the advice and services of her family dentist. He refused to extract the tooth while it was in "an abscessed condition," advising the patient to use hot packs to the face. This advice was followed at home until the patient became very ill and the family physician was called, under whose care she remained for three days. The following morning she was brought to the hospital by ambulance as a private patient. Her condition on admission was as follows: Temperature 103° F. The face was markedly swollen on the left side, the swelling extending down the neck and behind the ear. The scalp and forehead were edematous. Her left eye was closed and the lid puffed out to the level of the nose. The cheek was blue and pitted on pressure. The jaws were tightly closed, and the patient complained of pain in the small of the back, accompanied by a severe headache.

The operation consisted in free multiple incisions of the face and neck and the removal of the offending tooth. Drainage tubes were inserted in the neck and hot saline applications made. The saline applications were renewed every twenty minutes. Stimulation and continued enteroclysis was given. The anesthetic used was nitrous oxide and oxygen. For a while the patient seemed to do well, but relapsed during the night and died in the early morning.

CASE II.—*Sepsis following alveolar abscess, relieved by operation.* (Service of Dr. H. C. Deaver.)

The patient, female, aged forty-four years, was admitted on the same day as Case I. The diagnosis was the same with the exception that the abscess was on the right side of the face.

She first complained of pain in the lower right second molar. The following day her face began to swell. She used home remedies for two days and then consulted a dentist. He refused to extract the tooth while in an abscessed condition. On the fourth day she presented at the dental dispensary for treatment.

Condition on admission was as follows: The face was very much swollen on the left side, the swelling extending down the neck and behind the ear. The eye was closed and the lid puffed.

The face was just beginning to grow blue; at one spot it was very dark red and pitted slightly on pressure. The patient looked toxic, had the jaws held firmly together, and a temperature of 102.2° F.

Operation same as in Case I, and after treatment the same. In this case the anesthetic used was ether.

The patient was confined to bed for five days, during which time she was dressed twice daily. At the end of seven days she was discharged from the ward and referred back to the dispensary for further treatment. Was treated daily, and at the end of three weeks the incisions had healed entirely and the false ankylosis almost gone. When the patient was last seen, some two months after discharge from the dispensary, the result was most satisfactory, in that there was very little disfigurement of the face and the temporomandibular articulation was almost perfect. In both of these cases the pus obtained at time of operation was most vile.

Case III.—*Ludwig's angina following extraction of a tooth; operation; death.* (Service of Dr. C. C. Eves.)

The patient, male, aged nineteen years, had always enjoyed good health. He had a tooth extracted elsewhere under a local anesthetic. Several hours afterwards he suffered considerable pain in that socket. This gradually spread until his throat was quite painful on swallowing. It was at first thought when he applied at the hospital for treatment that he was suffering from a peritonsillar abscess on the right side, from which side the tooth had been extracted. The patient was admitted to the ward and several attempts to locate pus in the tonsils were made, but without success. Ice packs were applied externally and liquid food was given. The patient's temperature rose, and the following morning the floor of his mouth was edematous, his neck swollen, his respiration difficult, and an anxious look on his face. His breath was very offensive, and by the afternoon he had the symptoms of Ludwig's angina. Incisions were made in the neck along the median line up into the mouth, and in the submaxillary triangles connecting with the median incision. A slight amount of pus was found. Drainage tubes were inserted and flaxseed poultices applied continuously over the incisions. The ethyl chloride spray applied locally was used as an anesthetizing agent. Stimulants and continuous enteroclysis were given. The patient continued to grow worse; he had considerable difficulty in breathing, developed pneumonia, and died the following morning.

Case IV.—*Chancre of the gum.*

The patient, aged thirty-three years, father of three healthy children, had a carious tooth extracted at a "dental parlor."

About a month afterward he noticed his gum becoming sore over the socket of the extracted tooth. He sought relief at the same "dental parlor," but in vain, and consequently applied at the dispensary of this hospital. On examination the sore on his gum had the appearance of a chancre. The cervical glands were enlarged on the same side. A Wassermann test was made and found to be positive. The patient insisted that he had never had any venereal disease, hence it was natural to suppose that he had contracted syphilis in that dental parlor.

CASE V.—*Necrosis of mandible, following unrecognized alveolar abscess.* (Service of Dr. Neilson.)

The patient, a boy of eight years, developed an alveolar abscess on the lower left first molar. He was taken to a dentist, who refused to extract the tooth until the swelling had subsided. He, however, lanced the gum, but the pus was not in the gum tissue; it was in the jaw bone. The child had considerable pain, and the swelling in the neck growing larger, he was, on the following day, taken to a physician, who advised the use of flaxseed poultices externally. After the second poultice the abscess broke on the face, just below the mental foramen. The physician irrigated the newly formed sinus with hydrogen dioxide, and instructed the mother in the use of a small syringe charged with hydrogen dioxide with which the sinus was to be washed out three times a day.

Under this treatment, together with salves to heal the sinus, the child grew worse; so after three months the mother brought the boy to this hospital for treatment. The boy's condition on admission was as follows: The face was normal in size with a large indented area containing a suppurating sinus on the left side below the mental foramen. Examination with the aid of a probe revealed necrotic bone, which the *x*-ray later confirmed. The sinus discharged a considerable amount of pus daily. Intra-oral examination showed the teeth in a neglected condition and the lower left first molar badly broken down.

The operation consisted in opening, under ether anesthesia, along the border of the mandible and excising the necrotic bone, which extended back to the angle of the jaw and in extracting the decayed teeth present. The tissue was dissected well back so as to give a good flap to expose the area over the sinus, and the offending teeth were extracted.

The patient made an uneventful recovery, the wound healing rapidly. Two months later a plastic operation was done to improve the appearance of the patient. When seen several months afterward the result was most satisfactory.

Case VI.—*Necrosis of mandible following extraction.* (Service of Dr. T. R. Neilson.)

The patient, female, aged twenty-two years, had a carious lower left third molar extracted under local anesthesia. Her dentist prescribed as a mouth-wash hydrogen dioxide. As she had considerable pain in the socket, she returned to the dentist for relief. He irrigated the socket with hydrogen dioxide and instructed the patient to continue its use. Her face began to swell on the left side and her jaws became somewhat ankylosed. Two days later an osteopathic physician diagnosed her case as one of displacement of the cervical vertebræ and treated her accordingly, three times. She then consulted a physician, who advised and opened the swelling at the angle of the jaw, obtaining a free flow of pus. The pus continued to drain for three weeks, when it was decided to operate and curet the jaw. This was done, but without satisfactory result. A month later she was operated on again, still without result; and the pus continued to flow and became more vile in nature. The odor was so offensive that the patient refused to see any of her friends.

Condition on admission to this hospital was as follows: On the left side at the angle of the jaw a large sinus was present, surrounded by scar tissue and with an extremely offensive odor. The orifice of the sinus was about half an inch in diameter. The patient was anemic, and had some swelling present at the angle of the jaw. The flow of pus was profuse and yellowish-green in nature. Examination intraorally showed a small sinus in the region of the extracted third molar. On passing a probe into this sinus a distinct gritty feeling was transmitted through the probe to the hand. Examination through the external sinus gave the same sensation. *x*-ray examination showed a large sequestrum extending from the angle into the ramus.

The operation consisted in thoroughly opening the skin and dissecting back the muscles, exposing the necrotic area; this was removed *en masse*, and the surrounding bone well curetted and irrigated with normal saline solution. The wound was packed with iodoform gauze as drainage, and partly closed. Twice daily the wound was irrigated with a solution of:

| | | |
|---|---|---|
| Iodine crystals | | gr. 1 |
| Pot. iodid. | | gr. 15 |
| Zinc iodid. | | gr. 15 |
| Glycerin | | ʒ 4 |
| Water | q. s. ad | O 1 |

The patient was given food of strengthening nature, and when she was able to be out of bed kept out of doors. In three weeks' time she had regained nearly all of her lost vitality, and was brighter in appearance and disposition. The wound healed nicely, and at the end of five weeks had completely healed. Two months later a plastic operation was done and the scar-tissue removed, the result being most pleasing to both patient and operator.

CASE VII.—*Submaxillary abscess; impacted molar; reimplantation of an incisor.*

The patient, a man, aged twenty-three years, came to this hospital with a large swelling in the submaxillary area and the jaws tightly closed. He was unable to speak English, hence there was difficulty in obtaining a history. On pressure the swelling was exceedingly hard and painful, though no redness was present. A provisional diagnosis of a submaxillary abscess from an infected tooth was made. *x*-ray examination confirmed this diagnosis, showing the lower right third molar as the offending tooth. The third molar was lying in a more or less impacted position, butting against the second molar. The patient's temperature on admission was 102° F. The following day ether was administered, and the submaxillary area opened and drained by means of a rubber tube. Pus found was streptococcic and extremely foul in nature. In the attempt to open the mouth by means of a Mason gag for the purpose of removing the impacted and infected third molar, the left upper lateral incisor was completely dislodged from its socket. Owing to the great amount of trismus present, it was deemed advisable not to force the jaws apart too much, therefore the second molar was removed to give access to the deeply seated third molar, which was also removed. The pulp of the lateral incisor was removed through the foramen and the root canal filled; the tooth was then immersed in a 1 to 10,000 solution of mercury bichloride for a period of fifteen minutes, replaced in the socket and ligated with a silk ligature to the adjoining teeth twenty-three hours after its displacement. The cervical margin was painted with tincture of iodin twice daily for five days, when the ligatures were changed for a metal ligature such as is used in orthodontia. The wound in the submaxillary area responded quickly to treatment and left very little scar. In three weeks the ligatures on the lateral incisor were removed and the tooth was apparently solid in its socket. The patient was seen some five months later, when the lateral incisor was found to be doing excellent service and the site of operation on the neck to be barely showing.

Case VIII.—*Salivary fistula cured by operation.*

The patient, male aged forty-seven, presented himself at the dispensary with a parotid fistula. He had been struck in the left side of the face with a piece of metal, the face had immediately swelled, eye closed, and there was severe pain. He consulted a physician, who treated him by external applications for several days. The patient continued to have severe pains, especially during meal hours, so the attending physician decided to incise the face, and, in so doing, cut Stenson's duct, establishing a parotid fistula. Attempts at closure failed, and every time the patient smelled a savory dish he had had a profuse flow of saliva over his face.

The operation for closure of the wound consisted in making an artificial duct in the mouth just above the normal opening, and connecting this with the external opening. A fine rubber tube, attached to a probe, was drawn from the outside wound through the cheek into the mouth and secured to the mucous membrane. The external end was cut off short so as to be inside of the skin surface and was directed upward into the duct; the skin was then sutured with catgut and dressings applied. That night the patient suffered severe pains, and the face swelled up, almost closing his eye on the left side. In the late morning, however, the swelling subsided, and the saliva from the parotid gland commenced to flow through the rubber tube into the mouth. On the eighth day the sutures were removed from the face and the tube was removed from the inside of the mouth. The face healed well and the artificial duct continues to work satisfactorialy. The patient was seen two months later and expressed satisfaction, having had no recurrence of swelling or pain.

Case IX.—*Chronic inflammation of the submaxillary salivary gland, of dental origin.*

The patient a girl of twelve had a chronic enlargement of the submaxillary gland. On examination intra-orally her first permanent molars were found to be badly broken down. The root canals of the first permanent molar of the lower jaw contained pus. As the gland had been in a swollen condition for several months and had been treated externally without avail it was decided to remove it. The operation consisted of removal of the left submaxillary gland and the roots of first molar on the same side, also several other roots in the mouth. The socket of the left molar was well curretted. Upon examination after removal the gland showed a broken down caseous condition, but was not tuberculous. The patient made an uneventful recovery.

CASE X.—*Empyema of the maxillary antrum, followed by sarcoma.* (Service of Dr. H. C. Deaver.)

The patient, male, aged forty-three years, first noticed a soreness around the upper left first and second molars. Some days later abscesses formed around these teeth, and the patient had them extracted. Tenderness remained over the superior maxillary bone for some time. About two weeks prior to coming to the hospital a thick yellowish fluid was discharged from the left nostril. He was treated by a physician for this trouble. His face then began to appear swollen on the left side, but there was no further recurrence of pus discharged from the nose, two months after the first flow. The patient's condition on admission was as follows: He was a well-formed man, his face being somewhat fuller on the left side than on the right, and tender on deep pressure over the infra-orbital foramen. Examination inside the mouth showed the gum tissue to have healed where the molars had been extracted. The left side of the palate was somewhat bulged. $x$-ray examination revealed an abnormal condition of the antrum, but a decision as to whether it was an empyema or a malignant growth could not be made. The patient was etherized and the antrum opened through the mouth. On gaining entrance to this cavity it was found to contain pus. There was no sign of malignancy present, so the case was treated as one of empyema. About two weeks after operation it was noticed one day, while irrigating the sinus, that a growth had formed. This commenced to grow rapidly and a diagnosis of antral sarcoma was made. The operation, by Dr. Deaver, consisted in complete removal of the upper left jaw. The pathological report revealed spindle-celled sarcoma. In about two months it is hoped to construct an artificial jaw which will aid the patient in masticating and restore the contour of his face.

## SUMMARY OF CASES.

### From March, 1914, to April, 1915.

| | |
|---|---|
| Patients treated (including return visits) | 3654 |
| Extractions | 2319 |
| Extractions (impacted teeth) | 15 |
| General anesthetics administered | 932 |
| Local anesthetics administered | 128 |
| Permanent fillings inserted (alloys) | 410 |
| Guttapercha fillings inserted (mostly deciduous) | 919 |
| Gold fillings inserted | 4 |
| Gold inlays inserted | 9 |
| Artificial dentures | 4 |
| Porcelain crowns | 6 |
| Fractured jaws | 31 |
| Comminuted fractures | 3 |
| Oral surgical operations | 31 |
| Alveolar abscess (acute) | 221 |
| Alveolar abscess (chronic) | 210 |
| Syphilitic lesions of the mouth (secondaries) | 6 |
| Chancre of gums | 1 |
| Malignant lesions of mouth (including tongue, palate, cheek, mandible, antrum and maxilla) | 10 |
| Leucoplakia | 6 |
| Cervical adenitis cases | 30 |
| Enlarged submaxillary glands (dental origin) | 18 |
| Abscess of submaxillary region (dental origin) | 16 |
| Laceration of lips | 8 |
| Pyorrhea alveolaris (dispensary only) | 115 |
| Gingivitis cases (dispensary only) | 224 |
| Necrosis of jaws | 24 |
| Root amputation | 1 |
| Implantation of tooth | 1 |
| Trifacial neuralgia | 35 |
| Trifacial neuralgia (alcohol injections for same) | 4 |
| Stomatitis cases | 61 |
| Thrush cases | 2 |
| Epulis cases | 4 |
| Pathological dentition | 23 |
| Dentigerous cyst | 2 |
| Pulp stones | 15 |
| Supernumerary teeth | 18 |
| Fungoid pulps | 34 |
| Ranula | 2 |
| Ludwig's angina | 1 |
| Vincent's angina | 1 |
| Empyema of the antrum | 10 |
| Parotid abscess | 1 |
| Parotid fistula | 1 |
| Severe secondary hemorrhage | 12 |
| Case of hemophilia treated | 1 |

# ON EXCISION OF THE TONGUE[1]

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The purpose of this communication is to call attention to a method of excision of the tongue which may be described as a variant of Crespi and Bastianelli's modification of Langenbeck's operation. Crespi and Bastianelli's method is not described, so far as I can ascertain, in any of the text-books or systems of surgery at present in use in this country. I have merely mentioned it in my own text-book, and it is also mentioned, without credit to its originators, in Johnson's *Operative Therapeusis*, recently published.

Systematic excision of the tongue goes back to the time of Roux, who is credited[2] with having adopted in 1836 temporary division of the mandible; and who in 1839 did preliminary ligation of the lingual artery, finding then that after dividing the frænum and the anterior pillar of the fauces he could draw the tongue far outside the buccal cavity, and was able to complete the excision of its left half with facility. The method of operation popularized by Whitehead (1881) is a further development of that adopted by Roux.

Sédillot (1844) and Syme (1858) thought to make access easier by temporary division of the symphysis menti, and Kocher has now adopted a slight modification of this operation as his "normal procedure," and prefers it to the lateral suprahyoid operation formerly known by his name. The great advantage which median section of the mandible presents over Whitehead's method is that it permits simultaneous excision of the floor of the mouth when this is necessary. But as the carcinoma usually is situated at the side of the tongue, and spreads toward the fauces and pharynx, this approach gives insufficient access to the seat of disease in the majority of cases, and is best suited to those rather unusual cases where the carcinoma invades the mucosa around the frænum linguæ and does not extend far backward. Moreover, the wound left after operation by Sédillot's or Syme's technic is very prone to infection. In most cases, therefore, it is less applicable than Langenbeck's method (1875), which consists in making an incision from the angle of the mouth downward to the level of the thyroid cartilage, with division of the mandible between the first and second molar teeth. The remarkably low immediate mortality which attended this operation in Volkmann's clinic appears to have passed from memory as completely as the operation has from practice. In 1889 Krause reported Volkmann's results with this operation from 1875 to 1888: There were 35 such operations in which temporary resection of the jaw was done, with 2 deaths, a mortality of 5.7 per cent.; and there were 56 similar operations in which the disease was not so far advanced as to make division of the mandible necessary for complete extirpation, with not a single death from the operation; or, combining the two series of operations, there were 91 excisions of the tongue, with only 2 deaths, a mortality of 2.2 per cent., which compares favorably with much more modern statistics.

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[1] Read before the American Surgical Association, June 11, 1915; reprinted from Annals of Surgery, 1915, ii, 239.

[2] Demarquay makes this statement; and Verneuil refers to the "Roux-Sédillot Operation."

There are, however, two great objections to Langenbeck's method. The first is the unsightly scar, and the division of the branches of the facial nerve which the skin incision involves; the other, and a weightier objection, is that no adequate provision is made for extirpation of the involved lymph nodes. In Crespi and Bastianelli's modification (1889) of

Langenbeck's operation the former objection is absent, as the skin incision divides the lower lip in the midline and is continued outward along the body of the jaw to its angle and thence to the anterior border of the sternomastoid; but the second objection, the inadequacy of the exposure for extirpation of the lymphatics, still holds. They describe their method thus:

The soft parts are incised from the midline of the lower lip to the symphysis menti, and the incision is then prolonged outward along the lower border of the mandible to the anterior border of the sternomastoid. (In one case of invasion of the lateral wall of the pharynx, this incision was prolonged downward along the anterior border of the sternomastoid.)

This incision passes to bone through the lower lip and as far as the insertion of the masseter, and through the soft parts posteriorly as deep as necessary to allow the flap to be turned up well. This flap, composed of lower lip and cheek, is then turned upward. As the skin retracts downward, this exposes the submaxillary fossa, and the submaxillary salivary gland and lymph nodes can now be extirpated, and the lingual and facial arteries and their veins ligated. By virtue of the prolongation of the incision along the sternomastoid it is possible to expose the great vessels and to draw them out of harm's way by a retractor.

Then the mandible is divided. The best point is in front of the next to last molar tooth. The mandible is sawed obliquely, as recommended by Langenbeck, from behind outward and forward. The fragments now separate by muscular tension, and are pulled widely asunder, securing "enormous" space. Then it remains to remove the tumor, according to the individual case.

The mandible is sutured, and the flap (lower lip and cheek) is replaced and accurately sutured, with adequate provision for drainage.

In a later publication (1902) Bastianelli further extended this procedure, as follows:

By the incision represented in Fig. 124 *A*, he sought methodically the ablation of the lymphatics, even if they were apparently healthy, in all cases of lingual cancer, if the strength of the patient permitted.

A. *Neck Part of the Operation.* 1. First he makes a semilunar incision, with convexity below, from the symphysis menti along the midline to the hyoid bone, and then outward along its greater cornu, joining the anterior margin of the sternomastoid, and if necessary prolonged backward toward the nucha. The skin and connective tissue and platysma are incised, the external jugular vein is divided between two ligatures, and the flap is raised and fixed to the cheek by a suture. Then to clear the submental and submaxillary regions thoroughly the first step is to isolate and *ligate the external carotid artery* between the superior thyroid and lingual; or even just above the bifurcation. The venous branches must all be ligated doubly and cut between. Everything being cleared up to the mandible, the facial artery and vein are ligated here, and then the mass is removed. The branch of the facial nerve to the muscles of the lower lip is injured, but, as he points out, this is true of all operations.

2. Hemostasis being complete, the wound is tamponed, and one proceeds to the removal of the deep upper carotid glands and inferior cervical glands. The former are often invaded, and if they make a tumor it may be possible to remove them through the incision already made, dissecting them from the sternomastoid and perhaps from the pharyngeal wall, taking care to avoid injury to the superior laryngeal nerve. If they do not make a tumor, and are little enlarged and scattered, or if apparently healthy, it is necessary to use an additional incision to expose them. This *second incision* is made perpendicular to the first which it leaves at the anterior border of the sternomastoid, about the level of the hyoid bone, and passes obliquely back and out to the supraclavicular fossa (Fig. 124 *A*). In fact this incision runs almost parallel to the omohyoid muscle and a little higher. It may be neces-

sary to cut the sternomastoid across to facilitate exposure, turning its ends up and down and leaving its deep surface if necessary attached to the glandular mass. It may even be necessary to excise the muscle for a certain extent. He adopted section of the sternomastoid as a rule. The spinal accessory nerve is preserved. The dissection is difficult if there is no tumor mass, and numerous ligatures are required. The external jugular vein may be cut between two ligatures. The transverse scapular artery may be preserved. The cutaneous supraclavicular nerves are always cut, and the great vessels, the vagus and sympathetic nerves laid bare. The internal jugular vein may be resected with the glands if adherent. It is very important always to ligate or clamp above the tumor mass before cutting it out (to guard against recurrent bleeding). "The operation up to this stage always takes over one hour, good assistance, and patience."

If the other side of the neck is possibly involved, it is not operated on until the first side has healed. When the first side of the neck is completed, the wound is tamponed, and the second part of the operation (excision of the tongue) is undertaken immediately if the general condition of the patient permits; if not, it is postponed for eight to ten days. If a two-stage operation is necessary it is better to do the neck operation before that on the tongue; because, he says, after removal of the tongue there must elapse many days, sometimes *months*, before the patient can stand a second operation.

B. *Tongue Part of the Operation.* 1. If the lesion is limited to the anterior part of the tongue up to the pillar of the palate, or even if extensive but the tongue still mobile, one succeeds well by the intrabuccal method.

2. If the tumor, even if not diffuse, extends deeply toward the floor of the mouth, any intrabuccal operation is futile.

(*a*) If the tumor is mobile, even in part, Bastianelli follows Kocher's lateral suprahyoid method.

(*b*) If the tumor is diffuse, but not posteriorly, he adopts median division of the mandible.

(*c*) If the tongue is fixed and the tumor extends posteriorly, he adopts systematically division of the mandible near the next to last molar (turning the cheek aside as in Crespi and Bastianelli's method already described).

At the end of the operation Bastianelli sutures the inside of the mouth as well as possible, and then packs the mouth with iodoform gauze. The skin is sutured only at its three extremities: (1) in the midline, (2) at the posterior angle, and (3) below; thus leaving a great central opening which is packed with gauze to avoid secondary infection of the neck wound which he regards otherwise as inevitable.

The slight modification of Crespi and Bastianelli's operation, to which I desire to call attention, was worked out on the cadaver in the Laboratory of Operative Surgery of the University of Pennsylvania. The operation includes removal of the upper cervical and submaxillary lymphatics on the side affected, and of the tongue, at the same sitting. Thus it is designed for cases where the disease is undoubtedly still in the operable stage. As a dissection of this magnitude, when thoroughly done, requires two hours for its performance, very few patients will be in condition to endure a more extensive operation at one sitting. If a more extensive neck operation is required it will be proper in almost every case to divide the operation into two or more sittings. Whenever an operation for excision of the tongue is done in two or more stages, the surgeon should not neglect Bloodgood's sound advice to cauterize the floor of the mouth both from above and from below.

*Technique of the Operation.*[1] The patient being anesthetized[2] and placed in the head-high position, an incision is made from the point of the chin downward in the midline to the hyoid bone, and thence along the side of the neck affected, following the folds of the skin, well below the body of the mandible, to the mastoid (Fig. 117). This incision passes only as deep as the platysma, and is at once undermined beyond the midline in the submental region, and downward toward the root of the neck. When this undermining has been completed, a block dissection of the neck is commenced at the circumference of the area thus exposed. That is to say, the submental region is cleared down to the muscles (digastrics, mylohyoids, sternohyoids) from the far side of the midline toward the diseased side of the neck; and the dissection extends upward from well below the bifurcation of the common carotid artery to the floor of the mouth. As the dissection is carried from below upward the descendens hypoglossi nerve is spared if possible, but all venous branches and the superior thyroid, lingual, and facial arteries are doubly clamped, divided and ligated as they are encountered, thus ensuring the removal of all the lymphatics in one mass. The hypoglossal nerve itself is preserved, as well as the spinal accessory; both of them should be clearly exposed. The superior laryngeal nerve should also be preserved. The dissection is then continued across the mylohyoid and around its posterior border into the floor of the mouth, removing the entire submaxillary salivary gland along with the rest of the cervical tissues. The lower portion of the parotid gland is also cut away to ensure complete removal of lymphatics in this region. The mucosa between the tongue and mandible is not divided at this time, but the entire mass which has been dissected free is turned up with the skin flap upon the patient's cheek (Fig. 118). This mass is then dissected off the skin flap, the platysma being removed with the mass, and the facial artery being ligated on the body of the mandible, to prevent recurrent hemorrhage. The large wound is then swabbed out with a 2 per cent. alcoholic solution of iodin, and is covered with a hot, moist pack. This terminates the first stage of the operation, which consumes about an hour.

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[1] The figures illustrating the technique of the operation were drawn by Mr. E. F. Faber from dissections by the writer in the Laboratory of Operative Surgery of the University of Pennsylvania.

[2] Intratracheal insufflation of ether should be used, preceded by thirty minutes by a hypodermic injection of morphin sulphate (one-sixth of a grain) and atropin sulphate (one one-hundredth of a grain).

The *second stage* of the operation comprises the removal of the tongue. A stout linen suture is passed through the tip of the tongue, to be used as a tractor, and a mouth-gag is inserted on the opposite side of the mouth. The original skin incision is then continued upward from the point of the chin to the free border of the lip, dividing the lip in the midline. The mucous membrane between the lip and mandible is then divided with scissors, outward until the ascending ramus of the mandible is reached. The masseter is not cut. The cheek is then turned aside. This gives excellent access to the tongue. Another stout line suture is now passed through the glosso-epiglottidean fold, and both this and the suture through the tip of the tongue are drawn taut. Gauze is then packed loosely in the pharynx, around the intratracheal tube. The surgeon next divides the frænum linguæ with scissors, and continues the division of the mucous membrane of the floor of the mouth from this point backward to the anterior pillar of the fauces, first on the opposite, then on the diseased side. The anterior pillars of the fauces are then divided on both sides, and the tongue is drawn well out of the mouth (Fig. 119). The tongue is then severed half way across its base (at least 2 cm. beyond the visible limits of the carcinoma) on the diseased side, and then from the tip of the tongue backward along the floor of the mouth, to the transverse section, also on the diseased side. The bleeding up to this point will be trifling, as the lingual artery on this side has previously been tied at its origin in the neck (before the dorsalis linguæ branch is given off). The tongue is now held in the mouth solely by the hyoglossus and geniohyoglossus muscles on the side opposite to that diseased; as these are cut from before backward, the spurting lingual artery of that side is caught in a hemostat, and if the dorsalis linguæ also spurts it likewise is caught in the stump of the tongue, which is held up in good view by means of the suture previously passed through the glosso-epiglottidean fold. These clamped arteries are ligated. Molar teeth on the diseased side are then extracted and the

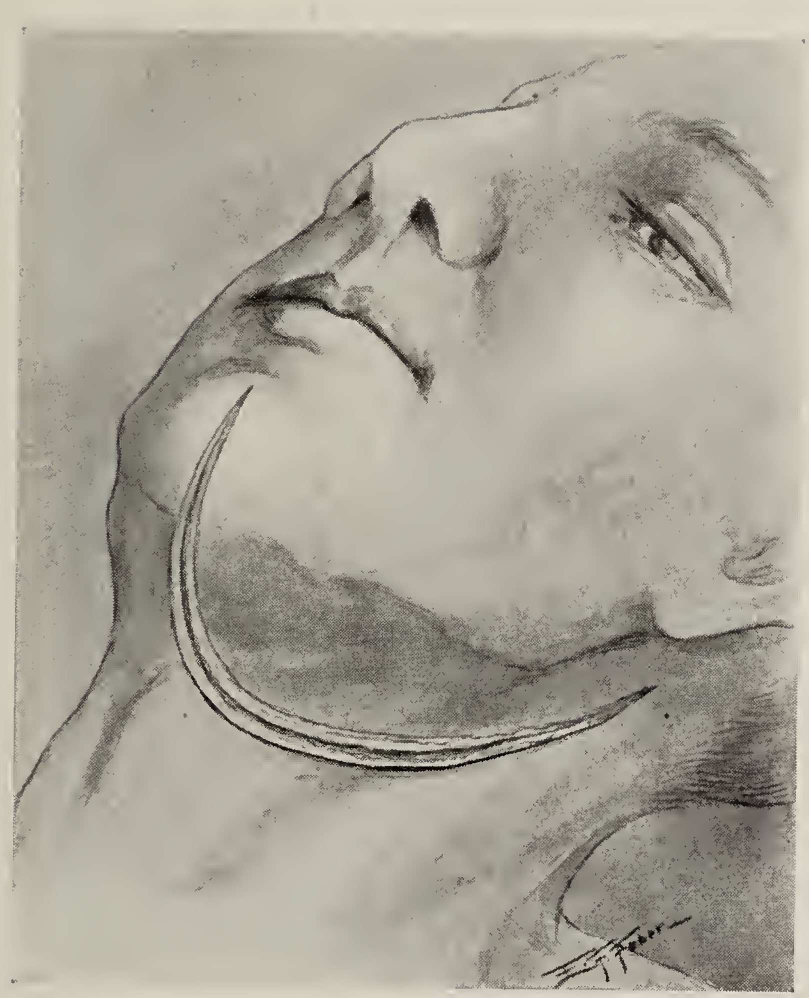


FIG. 117.—Skin incision for excision of the tongue.

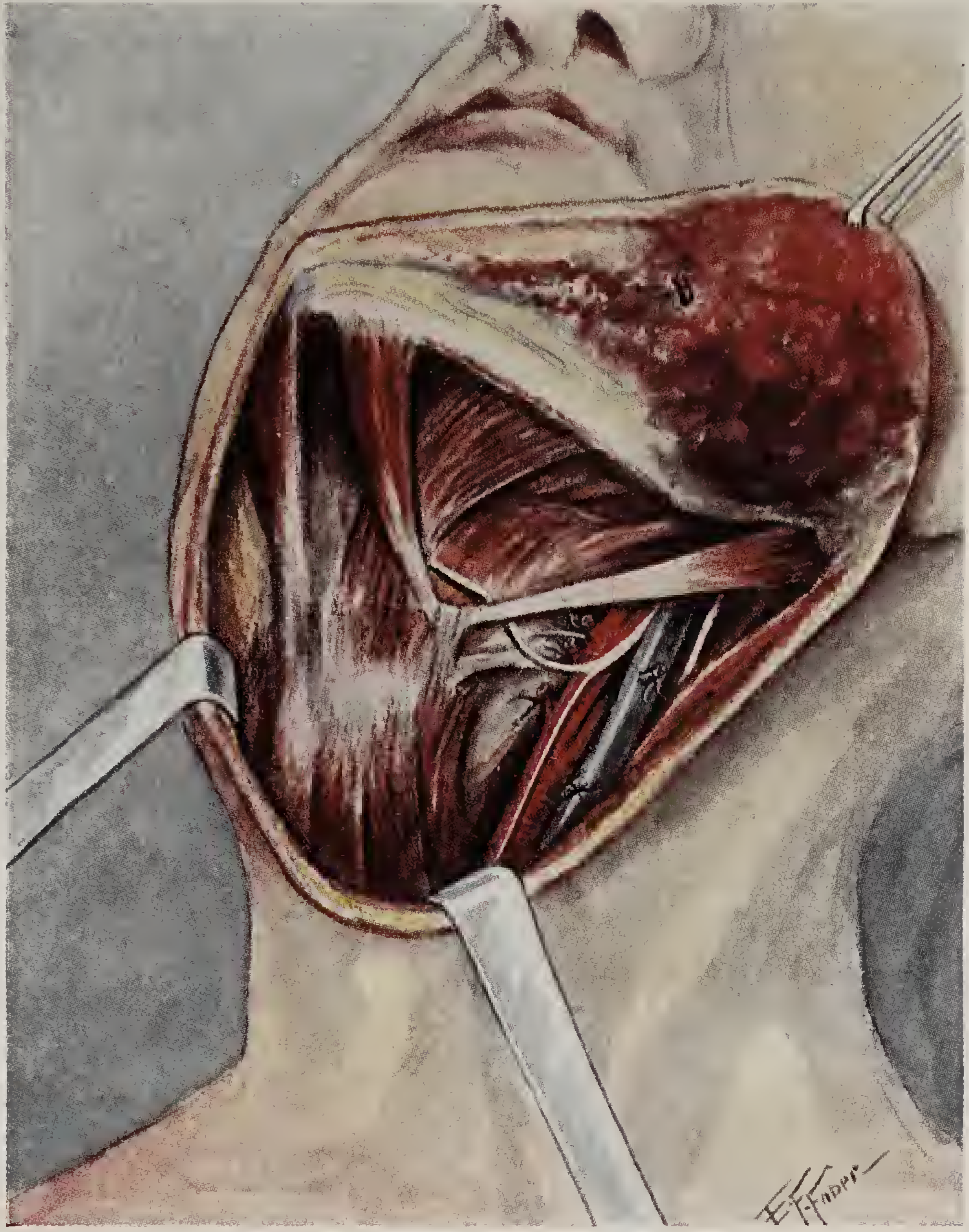


Fig. 118. The neck dissection has been completed, the cervical lymphatics and fat still attached to skin flap. The neck wound is then tamponed with gauze.

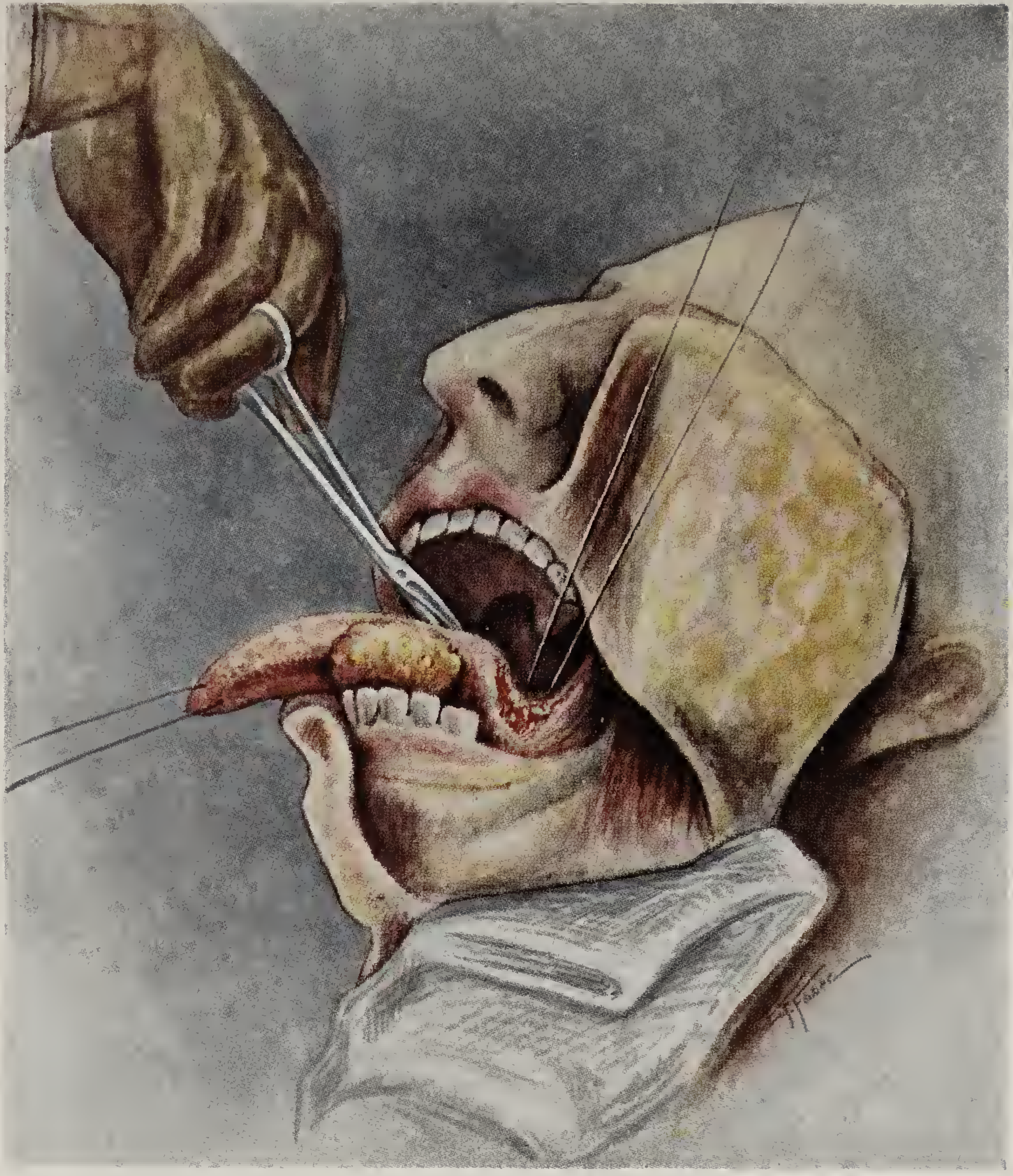


Fig. 119. The cheek has been turned aside, exposing the tongue. The left anterior pillar of the fauces has been divided, and the scissors are now dividing that on the right.

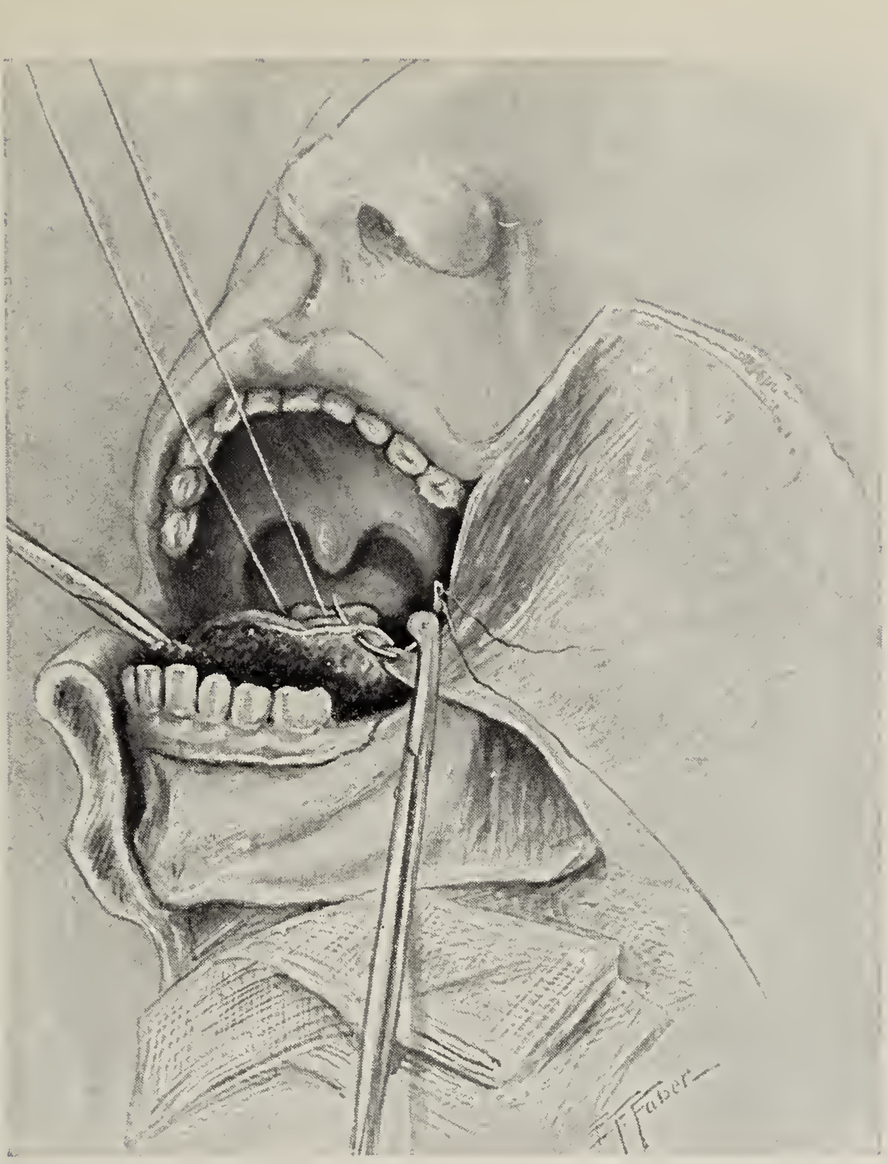


Fig. 120.—After removal of tongue, floor of mouth is covered partially by suturing mucosa of cheek across alveolus to stump of tongue. A hemostat is on the right lingual artery in the floor of the mouth.

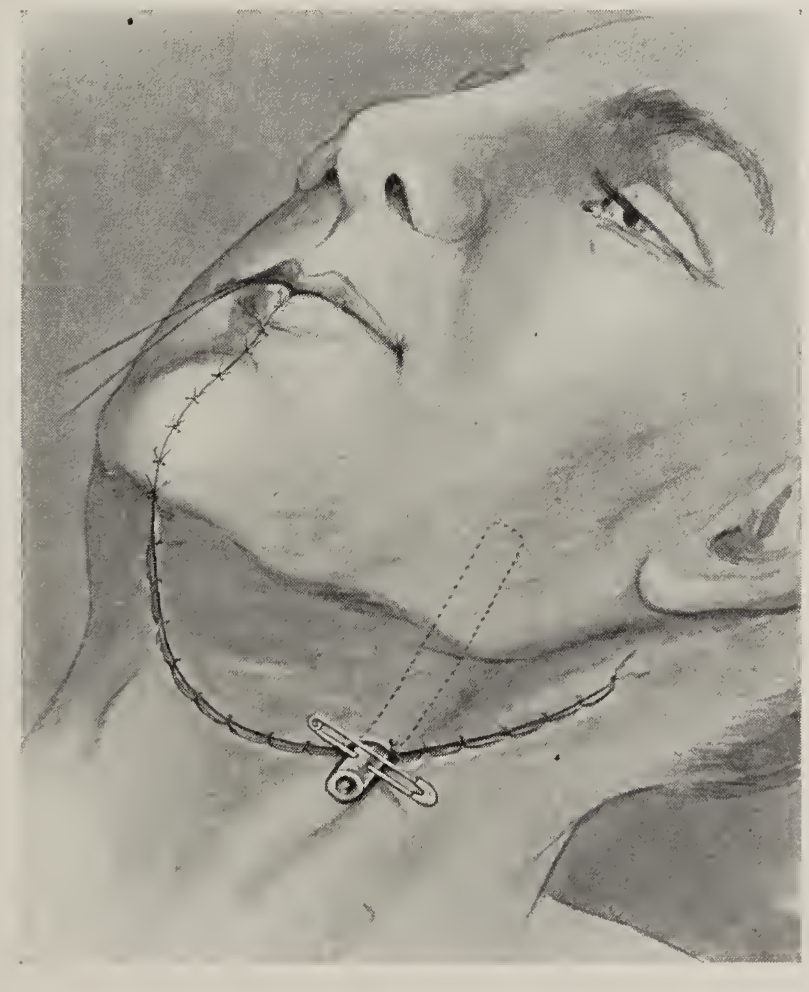


Fig. 121.—Skin incision sutured; drainage tube in place. Suture through glosso-epiglottidean fold retained for first 24 to 36 hours.

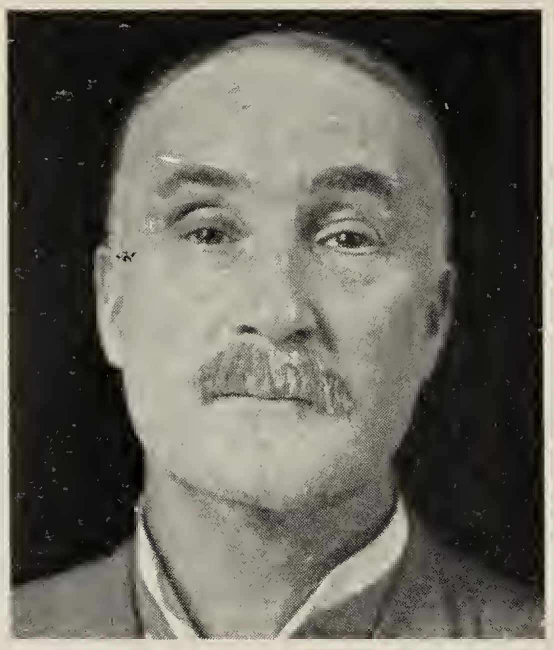


Fig. 122.—Excision of tongue, eight months after operation.

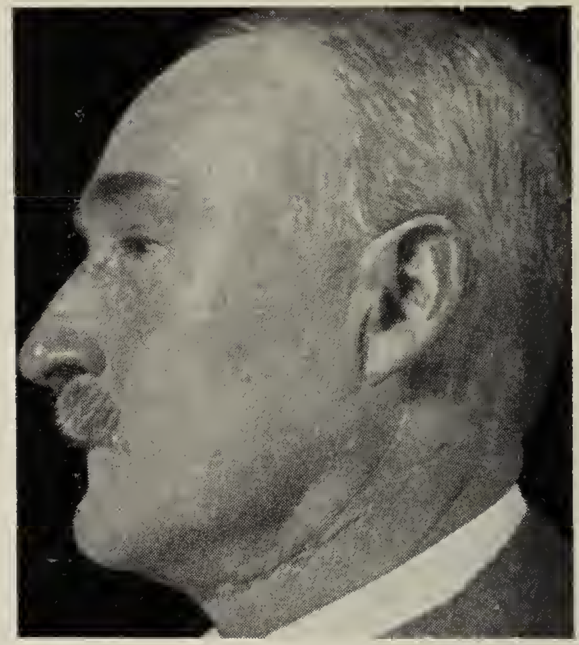
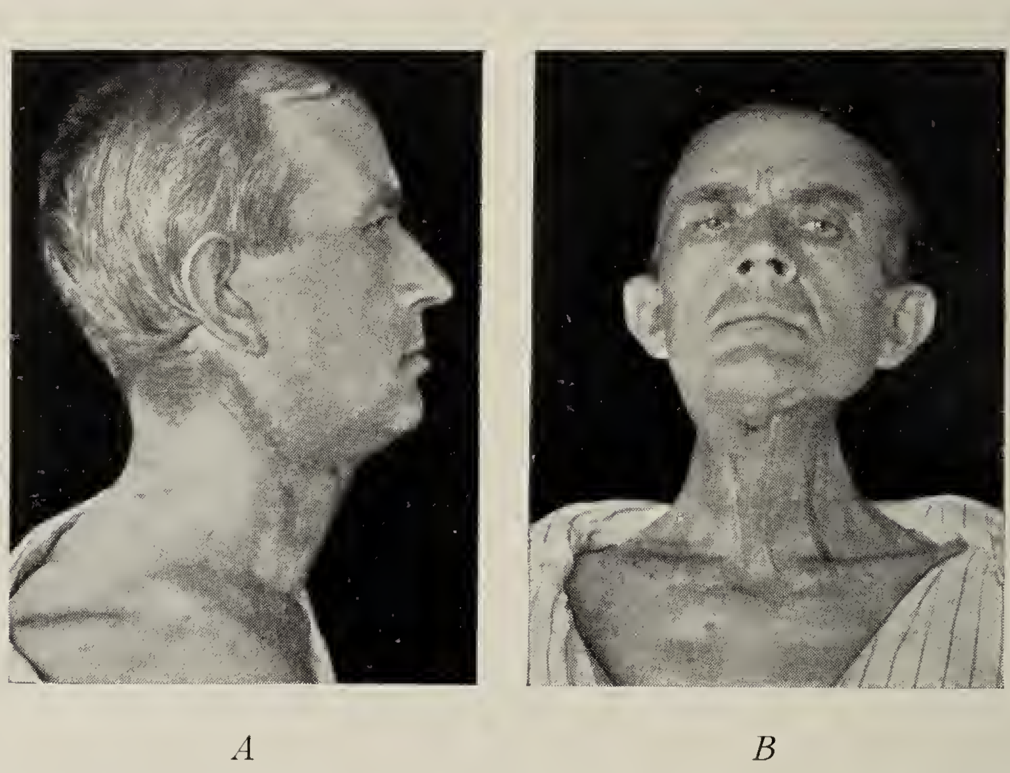


Fig. 123.—Excision of tongue, eight months after operation.



A

B

Fig. 125.—*A*, four weeks after second operation (on right side of neck); *B*, four weeks after second operation, two months after first operation (on left side of neck).

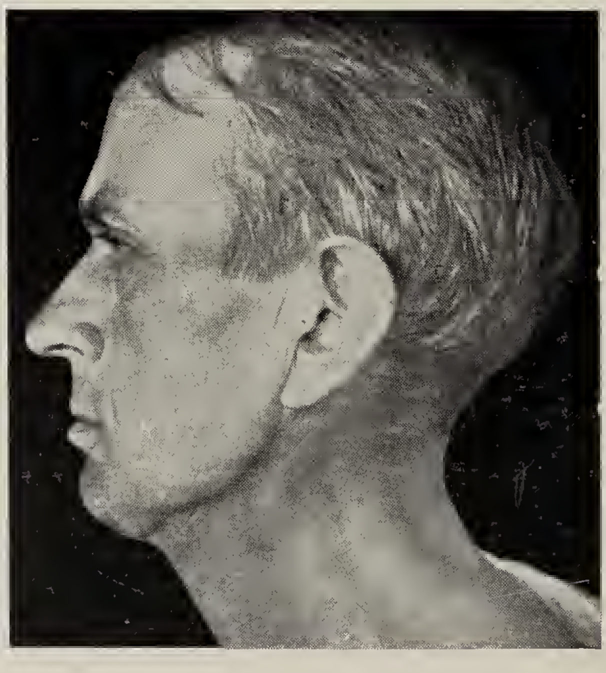


Fig. 126.—Two months after operation.

corresponding alveolus is cleared of mucous membrane; if necessary the alveolus may be excised. This permits the mucosa on the buccal surface of the cheek to be drawn in as a flap and sutured across the denuded alveolus to the stump of the tongue (Fig. 120), thus rendering a bucco-cervical fistula less likely. The remaining portion of the stump of the tongue is sutured as securely as is possible to the mucosa still remaining on the inner surface of the alveolus throughout its extent; and the mucosa of the vestibule of the mouth is sutured to that of the lower lip, from behind forward. A few buried sutures are used to attach the cheek to the body of the jaw, and the skin wound is accurately closed, with rubber tube drainage from below the floor of the mouth to the most dependent portion of the incision (Fig. 121). The pharyngeal gauze and intratracheal tube are then removed, but the suture through the glosso-epiglottidean fold is allowed to remain for twenty-four hours or longer, until the patient regains the power of swallowing.

A man, aged fifty-nine years, was operated on by me, at the Episcopal Hospital, by the method detailed above, on September 25, 1913, the carcinoma having been present for five months or more.[1] He was encouraged to swallow immediately after operation, and slept well the first night after swallowing a dose of paraldehyde. He was able to take liquid nourishment from the first attempt. On the third, fourth and fifth days after operation a little liquid food discharged through the neck wound in swallowing, but this did not recur. He left his bed on the sixth day, and walked out of the hospital on the twelfth day after operation.

The accompanying photographs (Figs. 122 and 123), made eight months after operation, show the excellent cosmetic result. At present, one year and eight months after operation, there is no evidence of recurrence, and the patient is in excellent health.[2]

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[1] This case was reported in Annals of Surgery, 1914, i, 289.

[2] This statement is still true when reading proof nearly three years after operation.

*Excision of All the Cervical Lymphatics.* When removal of all the cervical lypmphatics is contemplated it is advisable to do the excision of the tongue at a second operation, two or three weeks after the neck operation. I believe systematic excision of the sternomastoid and omohyoid muscles in one mass with the involved lymph nodes facilitates the dissection and renders recurrence less likely. I venture to suggest the following skin incisions for such an operation (Fig. 124 *B*).

The incision (*a-b*) begins at the hyoid *near* the midline, and passes straight downward to the suprasternal notch; it is continued thence along the clavicle to the acromion (*b-c*). The triangular flap thus outlined containing only the skin (not the platysma) is raised and the deep dissection is begun at the root of the neck after section of the sternal and clavicular attachments of the sternomastoid. When more room becomes necessary the incision is extended from *a* to *e*, and this large flap, *containing the skin only*, is reflected toward the trapezius as a base. When in the further course of the dissection the submaxillary and submental regions are reached, and still more room is required, the incision from *a* to *d* is made, and the flap *dae* is turned up over the mandible. In this manner free exposure of the entire side of the neck is

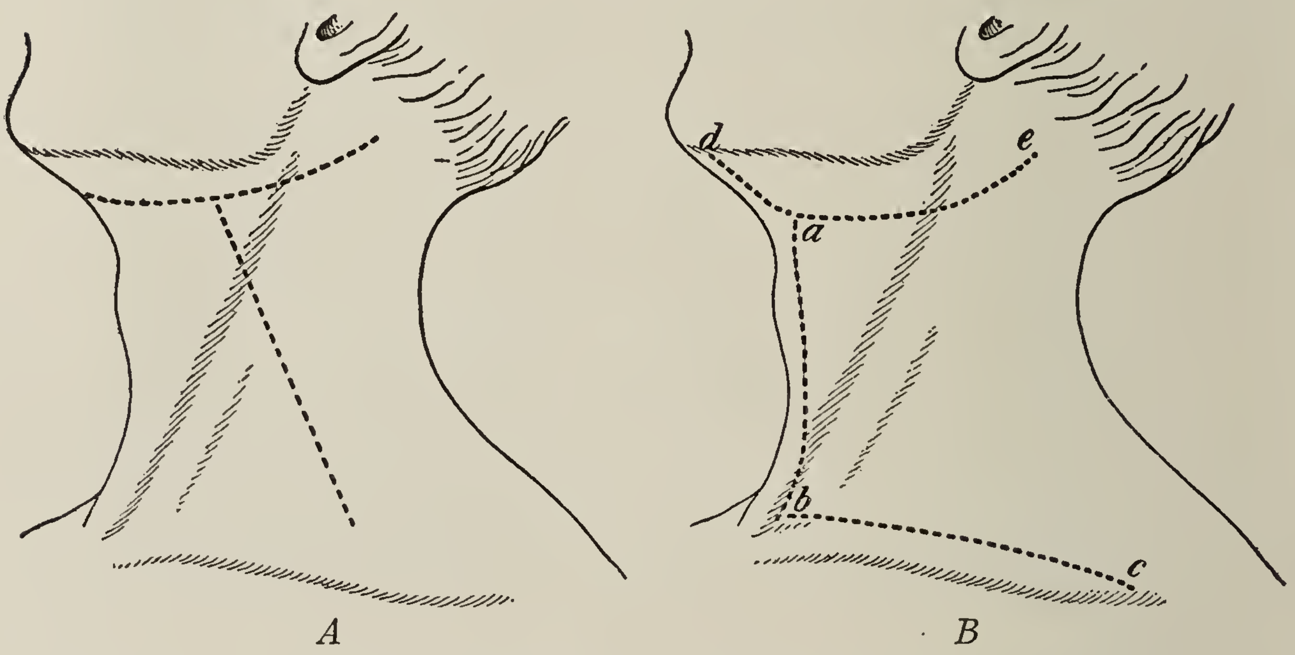

FIG. 124.—*A*, Bastianelli's incisions for extirpation of the cervical lymphatics; *B*, Incisions suggested by writer.

secured from clavicle to floor of mouth, and all the diseased structures are removed in one mass, the sternomastoid being sectioned again close to the mastoid. After thorough cauterization (black heat) of the floor of the mouth from beneath the jaw, the flaps are replaced and accurately sutured, with tube drainage at *a* and at *c*.

I had some fear at first that the extensive skin flap *eabc* might slough, but in the two instances in which I have employed this method (Figs. 125 and 126) the flap seemed to act as an immense Wolfe skin graft, and the only sloughing which occurred (insignificant in extent) was at the point *a*.

The object in making the incision *a-b* to one side of the midline is to provide for a similar incision a few weeks later on the other side of the neck; by leaving a small strip of skin in the midline between the two longitudinal incisions, better nutrition is assured the flap on the second side of the neck than if this were to be formed by cutting along the cicatrix of the first operation. As the flaps are well undermined in the submental region at each operation, no lymph nodes here can escape removal.

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# AFFECTIONS OF THE LINGUAL TONSIL AND THEIR TREATMENT[1]

By CHARLES C. BIEDERT, M.D.
AURIST AND LARYNGOLOGIST TO THE HOSPITAL

The so-called lingual tonsil is really not a tonsil at all, but simply a mass or collection of lymphoid tissue at the base of the tongue and in front of the epiglottis. This collection of lymphoid tissue varies greatly in size and shape, oftentimes being nothing more than a rather close aggregation of separate nodes, giving simply a roughened appearance to the base of the tongue. At other times it constitutes rather large masses which may be more or less pendulous.

The lingual tonsil forms the lowest portion of the lymphatic ring in the neck, known as the lymphatic ring of Waldeyer, the other portions of the ring being formed by the two faucial tonsils on either side and the pharyngeal tonsil above. Owing to the situation of the lingual tonsil and the difficulty of inspecting it, it not being seen upon the ordinary examination of the throat, the general practitioner is very likely to overlook affections of this tissue.

For the careful examination of the lingual tonsil it is necessary to use the head-light and the laryngeal mirror. I have frequently seen patients complaining of a sore throat, and upon the ordinary inspection of the throat nothing abnormal could be found, but by using the laryngeal mirror serious disease was found in the lingual tonsil, thus revealing the source of the soreness.

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[1] Read at the meeting of the Episcopal Hospital Clinical Society, Philadelphia, September 21, 1914. Reprint from the Laryngoscope, St. Louis, October, 1914.

The lingual tonsil is subject to all the inflammations and diseases that attack the other tonsils and tissues of the throat. I have frequently seen the extension of a follicular tonsillitis down to and involving the follicles of the lingual tonsil. We find simple inflammation and hypertrophy, lingual varix, tuberculosis, syphilis and malignant disease of the lingual tonsil; mycosis pharyngeus also frequently involves the lingual tonsil and various benign tumors have been observed. These conditions are all rather rare. I, myself, after rather a large experience in the examination of throats, have only seen one case of carcinoma of the lingual tonsil and probably six or eight cases of syphilis of the lingual tonsil. Syphilis is usually the tertiary manifestion or syphilitic gumma. Lingual varix is of frequent occurrence and often causes a great deal of trouble, producing an irritative cough, which after a time may cause one of the vessels to rupture, thus producing an alarming hemoptysis. This hard irritating cough of undemonstrable cause which lingual varix produces interferes with the rest of the patient, as a consequence of which he becomes very much run down and loses flesh, so that he and his friends fear tuberculosis of the lungs. I have on more than one occasion been able to demonstrate the source of the hemoptysis as coming from a ruptured vessel of the lingual tonsil and to assure the patient that his lungs were not diseased, and have by the proper treatment reduced the swelling of the lingual tonsil and eradicated the cough, and effected a complete recovery of the patient. Thus, in hemorrhage from the mouth or throat do not fail to make a careful examination of the lingual tonsil for the source of the bleeding.

Probably one of the most frequent affections of the lingual tonsil is simple hypertrophy, the symptoms of which vary. In some cases we will have a hard, dry, hacking, unproductive cough, especially worse at night or when the patient lies down. This cough may continue for months or years and be the only symptom complained of, as was the case in one patient who came under the writer's notice, a woman, who had a peculiar cough for twenty years by which she was known to all her friends. In this patient the cough was worse whenever she would get in a closed place, such as a theater or in church, and her advent into either of these places usually caused a procession of cough drops and cough lozenges to be passed to her from all directions by well-meaning and sympathizing people, until she finally had to remain away from public gatherings of all kinds. This cough of twenty years' standing was finally cured by the recognition and proper treatment of the cause—a simple hypertrophy of the lingual tonsil.

Cough is usually the predominant symptom in hypertrophy of the lingual tonsil, but in some cases we do not have any cough. The patients will complain of a sense of fullness in the throat or of a sensation of a lump in the throat, which they can neither swallow nor bring up (the so-called globus hystericus). Others complain of a tickling, prickling or burning sensation in the throat. It is surprising to what an extent these symptoms of simple hypertrophy will in some cases of nervous people be carried, as in one case which came to my notice, that of a nervous woman who thought she had some serious trouble with her throat and got herself worked up to a highly nervous state, in which she imagined that she could not swallow and that her throat was gradually closing, that she could not get her breath and that she was gradually choking to death. These symptoms were all relieved by the recognition of the cause, and by instituting the proper treatment for it.

Mycosis of the lingual tonsil usually occurs in association with mycosis of the pharnyx and tonsils. It causes a sensation of fullness, with tickling or prickling sensation in the throat. Upon examination we find the lingual tonsil dotted all over with whitish spots, somewhat raised from the surface and rather adherent, but they can be picked off and if placed under the microscope the diagnosis can be confirmed by finding the leptothrix buccalis, the causative fungi, one of the yeast fungi.

To recapitulate, then, we have the following diseases of the lingual tonsil in the order in which we find them most frequently: 1. A simple hypertrophy, causing slight symptoms of fullness and irritation in the throat, with cough. 2. A more or less greater enlargement in which the symptoms may be very pronounced. 3. Lingual varix, in which we have in addition to a hypertrophy of the tonsil, an enlargement and dilatation of the bloodvessels coursing over the tonsil. The vessels in some cases have been known to rupture and cause hemorrhage simulating hemorrhage from the lung. 4. The extension of a follicular tonsillitis to the follicles of the lingual tonsil, or in some cases where the faucial tonsils have been removed we often find a follicular lingual tonsillitis in association with a follicular pharyngitis. 5. Mycosis of the lingual tonsil, usually associated with mycosis of the pharnyx. 6. Syphilis of the lingual tonsil, usually gumma, although we do find mucous patches as an extension from the pharynx. 7. Tuberculosis of the lingual tonsil is rare, but is sometimes met with in advanced cases of tuberculosis of the lungs. 8. Malignant disease of the lingual tonsil is very uncommon, and, lastly, we may have any of the benign tumors affecting the lingual tonsil.

Diagnosis. The diagnosis of diseased conditions of the lingual tonsil cannot be made without the use of the head-mirror and laryngoscope. By having the patient grasp the tongue and pull it well forward we can then with the laryngoscope make a careful examination and arrive at the diagnosis.

Treatment. The treatment of lingual tonsil conditions is, as a rule, very simple. For the cases of simple hypertrophy, I have found that the application of a glycerole of iodin solution gives the best results. This is composed of iodin, 12 to 16 grains, and iodid of potassium, 24 to 32 grains to the ounce of glycerin. I apply this solution by means of a cotton swab. I have the patient pull the tongue well forward and am careful not to get any of the solution on the laryngeal side of the epiglottis. The U. S. P. official glycerole of tannin would be my second choice as an application in these conditions. Usually three or four applications of either of these remedies, made at intervals of two days, suffice to relieve the most obstinate cases. If it does not, it may then be necessary to cauterize the lingual tonsil, using trichloracetic acid, chromic acid, or preferably the galvano-cautery. In using these cauterizing agents great care must be exercised to limit the area cauterized to the lingual tonsil. In rare instances it may be necessary to remove a piece of the hypertrophied tonsil with the MacKenzie lingual tonsillotome.

The treatment of lingual varix is pretty much the same as that for simple hypertrophy. If we have one of the vessels bleeding it should be closed by searing it with the electric cautery.

In the treatment of mycosis of the lingual tonsil, I have found that applications of silver nitrate, 60 grains to the ounce, give the best results.

Syphilis, tuberculosis and malignant diseases of the lingual tonsil are best treated along the general lines laid down for the treatment of these diseases in any other part of the body.

# REPORT OF FOUR CASES OF LARYNGEAL COMPLICATIONS IN TYPHOID FEVER[1]

By CURTIS C. EVES, M.D.
AURIST AND LARYNGOLOGIST TO THE HOSPITAL

In these four cases observed during the past few months, three distinct types of laryngeal complications in typhoid are shown. That is: (*a*) laryngotyphoid, a rare onset in which typhoid fever is ushered in with an acute laryngitis; (*b*) laryngeal ulceration, in which ulcers are seen in the larynx simultaneously with those in the intestines; and (*c*) post-typhoidal perichondritis of the larynx coming on between the third and seventh week of the disease.

Case I.—A female, aged thirty years, came to the Episcopal Hospital complaining of sore throat, painful swallowing, dryness and hoarseness, with irritating cough, headache, abdominal pains, etc. She was admitted to the medical ward September, 1914. The second day after admission I examined her pharynx and larynx, which gave the appearance of a simple pharyngolaryngitis and acute inflammatory engorgement of the mucous membrane with scanty, sticky mucous exudate. Under local treatment the laryngeal condition improved. The temperature and general condition was not improving and a positive diagnosis of typhoid was made the second week after admission, at which time the pharynx and larynx had practically cleared.

This case coincides very closely with a rare form of onset in typhoid fever which Bayre, Lowrie, and Blum describe and term laryngotyphoid. It, however, may have been a coincident laryngitis happening with the onset of the typhoid.

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[1] Read before the Laryngological Section of the College of Physicians of Philadelphia, January 18, 1916.

In speaking of the onset of typhoid fever, Hare says: "A very rare respiratory disorder which may usher in typhoid fever, is that chain of symptoms known as laryngotyphoid, in which great hoarseness or aphonia develops with distinct evidence of acute laryngitis. These cases are quite different from those of severe ulcerating laryngitis seen in the advanced stages of the diesase."

Case II.—John P., aged twenty-one years; Irish; laborer; admitted to the Pennsylvania Hospital in August, 1915, suffering with typhoid fever. About twelve days after the attack began I was asked to see him in reference to his larynx. He complained of pain in the throat so severe that it kept him awake at night, also of pain on swallowing. There was a slight tenderness externally over the larynx. Upon internal examination of the larynx, five or six well-defined, elevated, grayish-white ulcers could be seen on the margin of the epiglottis, with one on the left ventricular band. There was a general redness of the pharynx and larynx with no noticeable edema. A 20 per cent. solution of argyrol was applied internally daily, with external applications of ice. Very little improvement in the symptoms or in the appearance of the condition was seen for ten days or two weeks, at which time with the subsidence of the typhoid, the ulcers gradually began to disappear. At the end of three weeks, the patient's larynx was in apparently normal condition.

This, I think, was a case of true typhoid ulceration of the larynx as described by Schultz, Wier, Jackson, Williams and others, some of whom were able to isolate the specific germ from sections of the larynx after death.

The ulcers disappeared as the fever and other symptoms subsided, and closely resembled in appearance the early stage of an ulcerating Peyer's patch.

Repeated attempts were made to obtain typhoid bacteria from the ulcers in this case, but without success; the prevailing organisms found were Staphylococcus albus and Streptococcus.

Case III.—Constineo C., male, aged thirty-five years; laborer; admitted to the ward of the Pennsylvania Hospital November 1915, suffering with typhoid fever. At the end of the fourth day I examined his larynx because of almost entire loss of voice. The patient had no difficulty in swallowing or breathing and complained of only slight pain upon pressure over the larynx. An examination showed two small ulcers on the posterior surface of the epiglottis near the margin. The cords were thickened and reddened without any apparent ulceration. This case was treated in the same manner as the second one. The ulcers disappeared with the fever, the hoarseness has gradually improved although it is not entirely gone at the present time, about three weeks since the fever has been practically normal. The patient is still at the Hospital. One week ago he had a slight relapse with a temperature of 100° F. for two days, but there was no reappearance of the laryngeal symptoms.

Case IV.—James A., Polish laborer, aged twenty-three years; admitted to the Episcopal Hospital November 10, 1913, suffering with typhoid fever. The disease ran a severe course with high temperature, intestinal hemorrhages, and profound toxemia. On the twenty-first day, when the disease was at its height, the patient developed an acute purulent otitis media with mastoiditis, followed in a few days by a subperiostal abscess, which was hurriedly opened, and some necrotic bone removed. The whole mastoid cavity had completely broken down in about three weeks from the onset of the otitis media purulenta acuta. During the whole course of the ear condition, the patient had complained of no pain, which was probably due to his toxic condition. The general condition of the patient rapidly improved after three weeks. After the mastoid was opened, there was free drainage of pus for four or five weeks after which time the cavity healed without further operation.

Three weeks after the mastoid was opened, that is, about three weeks after the temperature had been normal, during the seventh week of the patient's stay in the hospital, he was progressing satisfactorily; when a slight hoarseness of his voice was noticed during the afternoon of January 7, 1914. The hoarseness became rapidly worse; at seven o'clock slight dyspnea came on, the suffocation became more and more intense, with stertor, inspiratory depression of the neck and chest walls. The patient struggled desperately to get enough air through the larynx, the face became livid, and respiration very rapid. Intubation was tried three times by the Resident Physician but the tube was coughed out each time.

At this time the hospital located me out of town. I advised the resident to do an immediate tracheotomy, which he did at 11 P.M.

This relieved all struggle for air, and the patient was soon sleeping quietly. The temperature had risen to 101 ° F. and remained so for three days. Cold applications were made externally to the larynx and were kept up continually for two weeks. When I saw the patient the next day after the tracheotomy he had tenderness over the larynx externally and pain upon swallowing. Examination internally showed the laryngeal lumen entirely closed by edematous swelling of the epiglottis. The patient was kept quietly in bed on liquid diet, in a warm room where the air was kept slightly moist with steam. The patient complained of slight pain in the larynx, and there was a very moderate swelling of the tissues over the larynx, which was tender to the touch. On January 16, nine days after the laryngeal attack began, the edema of the epiglottis had slightly subsided, so that a small portion of the vocal cords could be seen. They were swollen so that the larynx was completely closed. The walls of the larynx were very much thickened, and the color of the mucous membrane was a darker red than at the onset.

On January 28, 1914, three weeks after tracheotomy, a direct laryngoscopic examination showed very little improvement. The mucous membrane was darker in color, with no opening into the larynx discernible. An 8 per cent. solution of silver nitrate was applied through the laryngoscope. Little improvement of the larynx was noticed during the next month, following local applications of silver nitrate solution.

On February 11, 1914, an attempt was made to introduce a large-sized intralaryngeal tube. The edema was still so great that in a few minutes it closed in over the top of the tube and the attempt was abandoned. Local applications were continued every second day. On March 6, the patient could first obtain some air through the larynx, when the tracheotomy tube was closed. From that time on the tube was closed for a short time each day. The length of time gradually increased until at the end of three months more the patient could breathe through the larynx with the tracheotomy tube closed for two hours at a time. He was then discharged from the hospital, having become adept at changing and sterilizing the tube. From this time on the patient visited the dispensary twice weekly, where applications of argyrol were made to the larynx. He was instructed to close the tube every day for two or three hours, which he claimed to do. The tube was taken out and the opening closed with a sterile cork for two hours at a time while the patient waited in the dispensary. After three months of treatment in the dispensary he could breathe for three hours with no apparent discomfort with the laryngeal wound closed. At the end of this time he was admitted to the hospital, where longer periods of breathing through the larynx were induced. For several days he went for four hours continuously twice daily without discomfort. He was then tried for a longer time; the opening was temporarily closed in the morning; at evening he was breathing so easily that it was decided to continue the laryngeal breathing throughout the night. About 10 P.M., he began to have difficulty getting his breath through the larynx, and in less than a half-hour the tube had to be hurriedly replaced. The next day the patient had a temperature of 102° F., with tenderness over the larynx. Internal examination showed a return of the edema although not so complete as at first.

After another period of rest of over six months the same treatment was tried of gradually lengthening the period of closure of the wound. However, we were never able to succeed in permanently closing the wound.

In July, 1915, the patient insisted upon returning to Poland owing to a family inheritance.

The cause of laryngeal ulcerations and perichondritis is thought by most writers to be due to infection of the tissues by the Eberth bacillus. Others believe that the friction of the surfaces caused by phonation, swallowing, and coughing upon tissues already suffering from typhoid toxemia is the most prominent causative factor. The first writers on the subject were of the opinion that the lesions were due to a secondary infection. Hare says "There can be no question that the specific organism of the disease is really accountable for the lesions."

Williams, Weil, Herbert, Liebman, and Jackson have obtained the typhoid bacillus from the lesions in the larynx after death of typhoid patients suffering with complications in the larynx. Specific infection, the toxemic state of the patient, and friction, together with secondary infection, no doubt are the leading factors in causing the complications.

Jackson believes that tracheotomy is the best treatment for the cases in which closure of the larynx takes place.

Writers who have studied this condition agree that the severity of the laryngeal lesions depends upon the severity of the typhoid infection.

Jackson found 68 cases of laryngeal involvement in a series of 360 cases of typhoid fever that he examined. He, with other writers on the subject, believes that entirely too few cases of laryngeal complications are recognized and that death occurs from this cause without laryngeal lesions being suspected in typhoid fever.

# GUNSHOT WOUNDS OF THE CHEST, WITH A REPORT OF TWO CASES

By JOHN PAUL JONES, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

Since 1906 there have been admitted to the hospital 16 cases of gunshot wounds of the chest; 9 of these cases recovered, while 7 were fatal, a mortality of 43.7 per cent. These were all penetrating wounds.

Gunshot wounds are divided into penetrating and non-penetrating: *Non-penetrating* (*a*), "Contusions, in which there is no laceration or penetrating of the skin, and (*b*), wounds including laceration or penetrating of the skin without involvement of the pleural cavity." (Lagarde.) Non-penetrating gunshot wounds in civil life deserve little consideration, as the missile inflicting the wound is small and the damage done is not serious.

*Penetrating* wounds of the chest, in the days of the old armament and in civil life, have a much greater mortality than in the wars of the present time, where only jacketed bullets are used. Otis records 8715 cases of penetrating wounds of the chest in the Civil War, with a mortality of 62.5 per cent. The French had a mortality of 91.6 per cent. in the Crimea, and the English 79.7 per cent. Of course, these cases would not receive immediate hospital treatment, as can the cases in the great cities. The mortality in modern warfare is very low as compared with the warfare when the old armament was in use and in civil life. Of 213 cases reported from the Spanish-American War and the Philippine insurrection our mortality was 24.5 per cent., as compared with 62.5 per cent. in the Civil War (Lagarde). Of 214 cases in the Boer War only 30 cases, or 14 per cent., were fatal (Stevenson).

The small frontage of the jacketed bullet and the minimum resistance of the lung tissue are conditions which cause the wounds to give very few symptoms when no other vital structures are damaged, or infection does not take place, and infection is rare with small missiles, except when the abdomen is invaded also.

Oettingen, from his observations in the Manchurian campaign, says when injuries of the lung are simple they are among the most harmless met with in war. Oftentimes there are practically no symptoms. After the battle of Santiago it was difficult to restrain the soldiers with penetrating wounds of the chest. Many had no dyspnea or hemoptysis (Lagarde). The results are often different in civil life when the soft-nosed bullet is necessarily deformed if it strikes any bone or other hard matter in its course. Wounds from unaltered small-bore bullets are seldom infected; those from bullets deformed by being ricochetted or large rifle bullets are usually septic (Spencer).

Symptoms. *Pain* is inconstant. Some patients complain severely while others can hardly be made to believe the seriousness of their condition.

*Hemoptysis* is present in 75 per cent. of the cases (Stevenson). It usually lasts from two to four days.

*Shock* is also variable. It is usually not pronounced unless there is marked injury to the chest wall, but this is by no means always so. In the case cited below there was extreme shock, the pulse could not be felt at the wrist, and we thought the patient could only live a short time, while there was only a small clean perforating wound in the second intercostal space.

*Cough* is usually slight and of short duration.

*Hemothorax* is common. According to Makins it occurs in 90 per cent. of the cases, and is almost always the result of injury to the intercostal vessels. The lung tissue is soft and contracted; usually only very small vessels are injured. The tissue contracts, closing the bleeding orifices. It is almost impossible to follow the tract of a small bullet wound through the pulmonary tissue on the postmortem table. The onset of hemothorax is gradual, usually not being demonstrable for two or three days. As the blood collects in the pleural cavity there is pain, cyanosis, dyspnea, restlessness, and rise of temperature. The fever is generally acknowledged to be due to absorption of fibrin and not to infection, though Spencer is inclined to the view of infection with the Staphylococcus albus, but usually no bacteria can be demonstrated.

*Empyema* increases in frequency with the caliber of the bullet and is usually due to foreign matter being carried in with the bullet. There is also often a secondary infection after aspiration or removal of a bullet.

*Pleurisy* is rare.

*Pneumonia* is seldom seen excepting in cases followed by shock or exposure. For the latter reason it is more common in military than in civil life.

*Abscess* of the lung is rare except when associated with the presence of a foreign body.

*Gangrene* of the lung is extremely rare. Two cases are reported by C. G. Spencer.

Treatment. The local treatment for these cases is simple. The area about the place of entrance or exit is painted with iodin and sterile dressings applied; in contrast to the treatment outlined by Guthrie one hundred years ago, who says, "The patient must be bled, vomited, purged, kept in the recumbent position, and cold applied so long as it shall be found agreeable to his feelings."

The patients should be partially propped up in bed and should not be moved until they are convalescent, as moving predisposes to hemothorax and empyema. In most cases properly treated the hemothorax is slight. The effused blood usually becomes absorbed if the patient is kept at rest and given sufficient morphin to keep him quiet. A hemothorax should not be aspirated unless it is great enough seriously to interfere with respiration, as it often is followed by empyema. The blood in the pleural cavity is an ideal pabulum for bacteria. This blood is usually sterile and is not apt to become infected for the smaller bronchioles and vesicular tissue of the lungs are for the most part sterile. Neither should lodged bullets be removed early unless they are causing symptoms. It is very tempting to remove a bullet when one feels it just beneath the skin. As a rule, its remaining there does no harm, while a great many cases of empyema are caused by removing superficially lodged bullets in the field hospital (Spencer).

The same author, in speaking of incision of the chest wall for removal of hemothorax, says it is unnecessary, unjustifiable, and dangerous.

Gunshot wounds of the heart, pericardium, through the root of the lung or through the lower chest, where the diaphragm and abdominal organs are involved, are much more dangerous than simple wounds of the lung and the mortality is extremely high.

Gunshot wounds of the heart are usually almost immediately fatal, but remarkable cases of recovery from these wounds are found in literature. Various reasons are given for these recoveries. Some observers claim that, owing to the heart and great vessels being held together by loose areolar tissue the heart is pushed aside by bullets with a low remaining velocity; it is also said that, owing to the volume of the heart being lower in systole, bullets might miss it then that would have penetrated it if the heart had been in diastole at the time. Mr. Makins thinks all penetrating wounds of the heart are fatal. Probably his views are not absolutely correct, as there are so many cases of recovery reported from bullet wounds of the heart inflicted by small bullets, especially the 6.5 mm. bullet of the Japanese.

Below are described the two last cases of gunshot wounds of the chest admitted to the hospital, both of which ran almost a typical course considering the location of the wounds. One was of the upper chest, which ran almost an uneventful course, with recovery, while the other was of the lower chest, which ran a stormy course and ended fatally.

Case I.—F. K., aged twenty-four years, strong Polish man, admitted to Dr. Frazier's service August 29, 1914. The patient attempted suicide, only a few moments before admission, with a 22-caliber pistol. On admission the shock was extreme. The radial pulse could not be felt at the wrist, and the heart rate was 160 per minute. His skin was cold and covered with perspiration. Examination revealed a very small round hole in the center of the second left interspace 5.6 cm. from the midline of the sternum. A little blood was oozing from the hole, and when he coughed a gush of air came from it. He did not expectorate any blood. The bullet was found just beneath the skin posteriorly over the seventh left rib 7 cm. from the spine. There was no discoloration of the skin at this point. Several rales were heard over the left lung and the breath sounds were faint, but there was no dulness and the lungs otherwise were clear. The heart action was very rapid and the sounds were distant. Nothing else abnormal was found in the physical examination. The patient soon reacted from the shock and his condition was good on the following day, except that he had a little fever.

During the first night he began expectorating a little blood, which continued for three days.

August 31. Continues to run a little fever. The left lung is dull to percussion below the third rib. No inspiratory sound can be heard and the expiratory sound is distant. The wound does not bleed and is healing. Air no longer comes out of the wound when he coughs.

September 2. Fever and flatness remain. I attempted aspiration but failed to recover any fluid.

September 10. The bullet was removed today by a small skin incision made under local anesthesia.

September 14. Dr. M. H. Fussell examined the patient today and reported the left pleural cavity full of fluid.

September 22. Flatness of the left chest remains and the temperature is a little higher. The patient evidently feels badly. The leucocytic count is 18,000, with 80 per cent. polymorphonuclears. We suspect empyema, and as the patient would not permit us to aspirate under local anesthesia, he is given ether and 4 cm. of the eighth left rib in the posterior axillary line is removed by Dr. A. P. C. Ashhurst. About one liter of bloody fluid is evacuated. The laboratory report shows the fluid to be sterile.

September 30. Fortunately empyema did not follow evacuation of the fluid, the patient's temperature is now normal and he is up and rapidly convalescing.

Case II.—A. G., a well-developed female, aged twenty-nine years. Patient was admitted to Dr. Mutschler's service on July 25, 1914, a short time after being shot three times with a revolver. On admission she is complaining bitterly of pain in the left chest. There is very little hemorrhage or shock.

The physical examination reveals a small round bullet wound over the seventh rib one-half inch from the margin of the sternum. In the region of the left tenth rib, in the posterior axillary line, is an area of swelling, ecchymosis, and tenderness. To percussion there is slight impairment of resonance at the base of the left lung, but the breath sounds are entirely normal. There is no hemoptysis. There is some rigidity of the abdominal muscles over the left hypochondrium. Peristalsis is normal. There is a second bullet wound just to the left of the third lumbar vertebra. The course of this bullet cannot be determined. There is a third bullet wound going laterally through the right arm anterior to the humerus. There is no evidence of division of any nerves or of the brachial artery.

July 28. Has run some fever ever since admission. There are signs of a collection of pus at the point of the lodgment of the chest bullet over the tenth left rib in the posterior axillary line. The area is incised and the bullet and quite a quantity of foul-smelling pus are removed. The abscess cavity is packed with gauze.

August 4. Patient continues to run fever, is restless at night, has a leucocytic count of 59,500, and the base of the left lung shows marked impairment of resonance, with very faint distant breath sounds.

August 6. As fever continues high, the left pleural cavity is aspirated today, but only bloody fluid was evacuated. The aspiration did not relieve the flatness, and a subphrenic abscess was suspected, so Dr. Richard S. Hooker was asked to examine the patient. His opinion coincided with that already entertained, that the patient was suffering from a subdiaphragmatic abscess. Dr. Ashhurst removed a portion of the tenth left rib near the angle under local anesthesia. This operation gave access to a large abscess cavity below the diaphragm, from which was evacuated a large quantity of pus with the odor characteristic of Bacillus coli communis. Drainage was instituted.

September 2. For the past three weeks the patient's temperature has been high and septic in character. The subdiaphragmatic abscess wound continues to drain profusely. The patient is evidently losing ground. Dr. Ashhurst again explored the chest today, as it is thought that she has a collection of pus which we have not found. A portion of the eighth left rib is resected anteriorly, and the diaphragm is found adherent to the chest wall. The diaphragm is incised and another pocket of pus from below the diaphragm is drained.

September 14. The subdiaphragmatic abscesses continue to drain. The patient is steadily growing weaker, her temperature is very irregular and she is delirious at times.

September 16. Patient died today. Unfortunately an autopsy was not permitted to show the exact course of the one bullet that caused all the symptoms, but the case goes to show how dangerous gunshot wounds of the chest are that also involve the subdiaphragmatic organs.

CONCLUSIONS. 1. Simple gunshot wounds of the lung are as a rule not dangerous.

2. Wounds below the 5th rib are very dangerous, as the diaphragm and abdominal organs are often involved.

3. Simple hemothorax should not be interfered with except when it is causing severe symptoms. Neither should lodged bullets in the chest wall be removed early, unless they are causing symptoms.

# GUNSHOT WOUNDS OF THE ABDOMEN, WITH A REPORT OF THREE CASES

By JOHN T. BENNETT, M.D.
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In the service of a large hospital a great many gunshot wounds are seen and treated. According to the records of the Episcopal Hospital, those of the abdomen rank fourth in point of number. I find that 41.2 per cent. are of the extremities, 20.2 per cent. head, 15 per cent. chest, 11.8 per cent. abdomen, 5.2 per cent. neck, 4.5 per cent. back, and 2.1 per cent. involve the chest and abdomen, or both together, with a limb.

Of the 20 gunshot wounds of the abdomen, 55 per cent. show no perforation of intra-abdominal structures, while 45 per cent. show injury to some of the organs. Of course practically all cases are operated upon, and of those showing perforations, there is a mortality of 66⅔ per cent. with 33⅓ per cent. recovery. Three of the 9 cases showing intra-abdominal injury had perforations of spleen, kidney, or liver; all of these died. Excluding this class of cases and counting only those 6 showing perforations of the intestinal tract, the recovery has been 50 per cent.

These figures are arrived at from the cases treated only within the past six or seven years, and would, of course, be somewhat altered if a more exhaustive search were made.

The following case histories are typical instances of abdominal gunshot wounds with perforation of the intestinal tract. Early operation (within a few hours of injury) almost always is successful, unless the lesions are so severe as to require intestinal resection, as in Case II. The other fatal cases were operated on 24 hours or more after injury.

Case I—.J. D., male, aged twenty-six years. (Dr. G. G. Davis's service, February 18, 1909.) While in a drunken brawl he was shot in the abdomen. When brought to the hospital he was under the influence of liquor, hence a clear history could not be obtained. He was not shocked; pulse rate, 80.

Examination showed a punctured wound on the upper margin of the umbilicus, through which a probe would follow two a one-half inches. There was no bleeding or escape of fecal material. No prolapse of intra-abdominal structures. No wound of exit.

The patient was operated upon three hours after the injury by Dr. Ashhurst. A median incision was made to the left of the linea alba at and below the umbilicus (six inches). When the puncture in the anterior sheath was found it easily admitted a hemostat into the peritoneal cavity. Upon opening the peritoneum, bloody fluid escaped. The small intestines were eventrated and two punctures in the ileum were found with extreme prolapse of the mucosa which blocked the escape of feces. The everting mucosa was cut away and the punctures closed with purse string sutures. The two punctures were then inverted together by continuous right angle linen suture, transverse to long axis of the bowel. Gas and fluid could just be made to pass the stricture thus formed. The stomach was intact, but there was found a hematoma in the gastrocolic omentum; this structure was opened and the lesser peritoneal cavity explored which was found to be dry. Opening in the gastrocolic omentum was then closed. A bruised area on a section of small intestine was inverted. The peritoneal cavity was then irrigated with large quantities of salt solution which could not be made to return clear. A further exploration revealed a retroperitoneal hematoma along the right common iliac vessels. A wound was found in the posterior parietal peritoneum to the right of the third or fourth lumbar vertebra below insertion of mesentary and just to the left of the cecum. This was enlarged and no active bleeding found. It was then packed with gauze. A glass tube was placed in the pelvis and the abdominal incision was closed with through-and-through silkworm-gut sutures. Anterior sheath approximated with interrupted chromic catgut. The glass tube and the pack emerged from lower angle of incision. The patient reacted well from the operation. He was placed in the Fowler position and was given continuous enteroclysis. He was kept quiet with small frequent doses of morphin. The glass tube was exhausted twice daily and removed entirely on the second day. Forty-eight hours after the operation small quantities of liquid food were given by mouth. On the third day the bowels were well opened with enemata and on the fifth day the gauze packing was removed which had a fecal odor. The stitches were removed on the tenth day and at this time there was a pronounced fecal fistula but no prolapse of intestine. The patient was at this time placed on solid food and in three days the fecal discharge had ceased. The sinus then closed uninterruptedly and patient was discharged cured March 18, 1909.

Case II.—G. F., male, aged twenty-two years. (Dr. Frazier's service, November 12, 1907.) Brought to the hospital one-half hour after shooting himself in the abdomen.

On admission: Patient very much excited, but not especially shocked. Abdomen showed a punctured wound two inches below the right costal margin in the mammary line. No point of exit. Abdomen was rigid, tender, and showed oozing of blood from the puncture, but no fecal material. He complained of intense pain in the abdomen. The urine did not show blood.

Patient was operated upon by Dr. A. P. C. Ashhurst one and three-quarter hours after the injury. A long right rectus incision was made, and upon opening the peritoneum, blood-tinged fluid and gas escaped. Six perforations of the small bowel and one of the ascending colon were found. The colon was closed with purse-string suture. The punctures in the small bowel were so close together that resection of four inches was required and an end-to-end anastomosis was done. Visible fecal matter was wiped out, and a gauze drain was placed in the rectus incision, which was then closed with through-and-through silkworm-gut sutures. A suprapubic puncture was then made and a glass tube inserted into the pelvis for drainage.

Patient reacted well from the operation. He was placed in the Fowler position with continuous proctoclysis. His condition seemed fairly good until the second day, when he began to grow weaker, and died of peritoneal sepsis.

Case III.—Francis B., male, aged eight years. (Dr. Ashhurst's service, January 9, 1916.) Accidently shot in the abdomen with a 22-calibre rifle. Brought to the hospital about one hour after the injury.

On admission: Well-developed white boy with no evidence of shock, but he complained of severe pain in the abdomen. The face was flushed, pulse not unduly rapid, respirations slow but entirely thoracic. Abdominal wall showed a small punctured wound one inch to the right of and one-half inch below the umbilicus. The abdomen was slightly rigid, but very tender and peristalsis could be heard occasionally. There was no nausea or vomiting. Bowels had not moved since the injury and urine was negative for blood. No wound of exit.

Patient was operated upon by Dr. Ashhurst two hours after admission (about three hours after the injury). A right rectus incision was made. Upon opening the peritoneum, bloody fluid and gas escaped. Scattered through a space of twelve inches there were found seven punctures of the small intestine. The mucosa was protruding and blocked the passages. The perforations were closed with purse-string sutures of linen. A glass tube and cigarette drain were placed in the pelvis and the incision closed with through-and-through silkworm-gut stitches, the drainage tube and wick emerging from the lower angle of the incision.

The patient reacted well from the operation. He was placed in the Fowler position with continuous enteroclysis and small doses of morphin were given sufficient to keep him quiet. The glass tube was exhausted twice daily and was removed on the second day when small quantities of water were given by mouth. On the third day he was given small quantities of broth and albumen, increasing the amount each day. He vomited greenish material a few times on the fourth day and peristalsis became active, leading to the suspicion of intestinal obstruction. This symptom gradually subsided and on the sixth day he was placed upon full liquid diet. The distension that occurred all along was relieved with enemata. The temperature did not at any time reach above 101.3° F., and it remained normal after the fourth day.

The stitches were removed on the eleventh day. The incision closed at the end of three weeks; there was at no time any purulent or fecal discharge. He was allowed to get out of bed on the eighteenth day, and was discharged cured on the thirtieth day.

# THE HYPERCHLORHYDRIC SYNDROME: PATHOGENESIS AND TREATMENT[1]

By A. A. STEVENS, M.D.
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Although hyperchlorhydria has been the subject of numerous investigations, and there is already an enormous amount of literature bearing upon it, there are still many points relating to the condition which require elucidation. Hydrochloric acid is secreted probably only in the fundus of the stomach, and the quantity produced is influenced by a large number of factors. Some of these operate in a reflex way, the vagi serving to transmit to the stomach the impulses which have been excited in the central nervous system, while others operate directly upon the stomach itself, or, as is generally assumed, upon the sympathetic endings in the wall of the stomach. That these two forms of secretion—the cephalogenous and the endogenous—exist can readily be demonstrated experimentally. Thus food that is masticated and swallowed, but is allowed to escape through an esophageal fistula before it reaches the stomach, excites a flow of normal gastric juice; and, on the other hand, food that is introduced directly into the stomach through a gastric fistula and which has not been seen, touched, smelled, tasted, or masticated, also calls forth normal gastric juice. The chief factors in reflex or cephalogenous secretion are the appetite, the sensations of touch, taste, smell and sight, and probably the act of mastication itself. The important factors in endogenous secretion are the physical state of the food, its temperature, and, above all, its chemical composition. The total secretion of hydrochloric acid, unlike the total secretion of gastric juice, is not related to the amount of food ingested, but is approximately proportional to the protein in the diet, the physical state of the food and the temperature of the food being factors of minor importance. With meats the secretion is the most acid, and with carbohydrates it is the least acid. Fats depress acid secretion. Normally, whatever the diet there is a tendency for the establishment of a fairly constant degree of free acidity, although the quantity fluctuates within narrow boundaries. In certain pathologic conditions, however, the total amount of acid secreted is far more than is necessary to satisfy the affinities of the protein in the stomach, both that taken as food and that contained in the mucus, and in consequence the excess of free hydrochloric acid rises above the normal.

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[1] Reprinted from the Therapeutic Gazette, August, 1913.

As to what constitutes hyperacidity, opinions differ. Considering the matter from the purely chemical standpoint, however, the majority of writers assume that any amount above 0.2 per cent. is to be regarded as a hyperacidity. The question of the relative frequency of the condition based upon definite percentages has also been variously answered by different observers. Fleiner, Westphalen, and others have claimed that it is comparatively rare, while Bouveret, Riegel, and many American authorities believe that it is very common, not a few of the latter estimating the frequency at 50 per cent. of all chronic gastric affections. This difference of opinion as to the comparative frequency of hyperchlorhydria, while it may be ascribed in part to differences in nationality and social conditions, is due very largely, without doubt, to a lack of uniformity in the methods of examination. Some investigators have based their calculations upon one or at most two examinations, which cannot be regarded as sufficient. The findings in first examinations are often misleading, as the emotional disturbance excited by the use of the stomach tube frequently causes temporary derangement of secretion, and, moreover, in many individuals the amount of acid secreted varies considerably from day to day. It is not an uncommon experience to find on one day hypochlorhydria and on the succeeding day well-marked hyperchlorhydria. Then, again, some of the studies have been made upon private cases, while others have been made in hospitals. The results have naturally been divergent, for, as Riegel has said, hyperchlorhydria is more likely to be seen in private practice than in hospital practice, the symptoms of the condition rarely becoming so severe that patients are forced to enter a hospital. Finally, some experimenters have worked with the non-stimulating trial breakfast of Ewald, and others have employed the more palatable proteid-containing meal of Riegel. The lesson to be drawn from all this, and which seems to me should be given more emphasis, is that the results obtained from chemical analysis of the stomach contents are of little aid in diagnosis, unless they are based upon several examinations with different meals, and unless they show pronounced departures from the standard usually accepted as normal, as the amount and quality of the secretion depend upon so many factors and the variations seen in the same individual under different circumstances are so great.

Another factor which adds even more to the difficulty of determining the relative frequency of hyperchlorhydria is the total lack of harmony in many cases between the chemical findings and the subjective symptoms. Clinically, three groups of cases may be recognized: First, those in which the usual subjective disturbances of hyperchlorhydria are present and examination of the gastric contents reveals a high degree of acidity—60 or over; secondly, cases in which the subjective disturbances of hyperchlorhydria are present but in which examination of the gastric contents reveals moderate or even low acidity; thirdly, cases in which no gastric symptoms are present, and yet examination of the gastric contents reveals a high degree of acidity. Stockton (*Journal of the American Medical Association*, January 11, 1902) was the first to point out that in many cases in which the symptoms of hyperchlorhydria are present the acidity is really normal or subnormal, and this view was subsequently supported by Steele, von Noorden, and Riegel. Laboulais had under observation a patient with symptoms of hyperchlorhydria, and proved by analysis of the gastric contents that at the very time when the pains were relieved by the administration of sodium bicarbonate there was practically no hydrochloric acid present in the stomach.

On the other hand, Schüle (*Zeitsch. f. klin. Med.*, 1895, xxviii), Sorensen and Brandenburg (*Archiv f. Verdauungskrank.*, 1897, iii), Gintls (*Archiv f. Verdauungskrank.*, 1898, iv), Illoway (*Archiv f. Verdauungskrank.*, 1902, viii), Kauffmann (*Zeitsch. f. klin. Med.*, 1905, viii), Steele (*Jour. Amer. Med. Assoc.*, August 18, 1906), and others have cited cases showing that a great excess of hydrochloric acid may exist without any symptoms at all. Kauffmann in a series of 19 patients free from gastric symptoms found a total acidity of over 70 in ten and over 100 in two. Herschell (*Brit. Med. Jour.*, January 21, 1911) in 100 consecutive cases presenting the symptoms of hyperchlorhydria found an excess of acid in 68, normal acidity in 21, and subacidity in 11.

It is evident therefore, that an excess of hydrochloric acid is not the sole factor in producing the hyperchlorhydric syndrome, otherwise the latter would be present in all cases of high acidity and absent in all cases of low acidity. In further confirmation of this view is the fact that the symptoms of hyperchlorhydria often disappear under appropriate treatment even though the degree of acidity still remains high, and also the fact that the introduction of hydrochloric acid into the stomach in amounts much in excess of those ever found in disease is not productive of pain. In three patients on whom gastrostomy had been peformed Schmidt (*Mitt. a. d. Grenz. geb. d. Med. u. Chir.*, 1908, xix) introduced into the stomach from 20 to 40 c.c. of ½ and 1 per cent. solutions of hydrochloric acid, without exciting the slightest pain or discomfort. Two of the patients subsequently died, and autopsy revealed perfectly normal gastric mucosæ. Hertz (*Lancet*, April 22, 1911) has repeated these experiments with the same results. In six cases of gastric ulcer, in all of which the diagnosis was confirmed by operation, 4 ounces of 0.5 per cent. solution of hydrochloric acid, introduced into the stomach by a tube, produced no sensation whatever. It is thus clear that the pain which is often associated with an excess of hydrochloric acid in the stomach cannot be due to the action of the acid alone. It is equally clear, however, that the acid is in some way connected with the production of pain, as rapid relief always follows the administration of an alkali or the evacuation of the stomach.

It is of the utmost importance to determine, if possible, what the factor is which in conjunction with an excess of acid, and sometimes in the absence of such excess, is the cause of abdominal pain, for treatment that does not take this factor into consideration can scarcely be more than symptomatic.

Talma (*Zeitsch. f. klin. Med.*, 1884, Bd. xiii) suggested that the symptoms of hyperchlorhydria may be due to hyperesthesia of the gastric mucosa, and this view has been accepted by Stockton, Steele, Kauffmann, and others. However, the histologic studies of Gaskell, Langley, and others, and the investigations of Lennander, James Mackenzie, and more recently of Hertz (Goulstonian Lectures on the Sensibility of the Alimentary Canal, 1911) have demonstrated conclusively that the stomach and other viscera are not sensitive to tactile or painful stimulation, and that the afferent nerves of most organs convey impulses without sensation of any kind being experienced. It is generally accepted. therefore, that pain which feels as if located in the stomach has its true seat in one or other of the structures of the abdominal wall, the afferent sympathetic nerves conveying certain stimuli originating in the stomach to the segments of the spinal cord from which come the sensory nerves supplying the skin and muscles in the painful region. If we accept this view, we can readily understand why the tender area in gastric ulcer may be quite remote from the actual seat of the ulcer unless it is caused directly by the spread of the inflammation to the sensitive overlying structures.

The explanation of the pain in hyperchlorhydria, as well as of that in gastric and duodenal ulcers, which best harmonizes with all the facts, is that it is the peripheral expression of a cramp or spasm affecting the unstriped muscular fibers in the stomach, and, therefore, is analogous to that produced by irregular or excessive contraction of unstriped muscle in other organs, as for instance the bowel in cases of mucous colic, an affection in which pain is also combined with secretory perversion. Modern experimental investigation strongly suggests that abnormal excitability of the vagus region may be a factor, at least in some cases, in initiating the painful spasm which appears in the hyperchlorhydric syndrome, as well as in many cases of peptic ulcer, and that it may in some instances even play a part in the production of gastric erosion and ulceration. The results of numerous experiments show conclusively that the sphincters of the stomach are partially controlled by the central nervous system, and that in general the fibers received from the vagus are augmenters. Artificial stimulation of them causes, as May (*Jour. of Physiol.*, 1904, xxx) has shown, first, an inhibition of the peristalsis of the pyloric portion, with a lessened tone of the cardia, followed later by an acceleration of peristalsis in the pyloric region with increased tone in the cardia. Talma (*Zeitsch. f. klin. Med.*, 1890, xvii) faradized the vagus and caused spasmodic contractions of the stomach. He then applied dilute hydrochloric acid to the mucosa and produced gastric ulcers. Van Yzeren (*Zeitsch. f. klin. Med.*, 1901, xliii) and Ophüls (*Jour. Exp. Med.*, 1906, viii) got similar results in rabbits by cutting the vagi below the diaphragm, although according to the former the vagotomy caused cramp of the stomach, and according to the latter it caused marked dilatation. Westphal quoted by Von Bergmann (*Münch. med. Woch.*, 1913, lx) obtained hemorrhagic erosions in the stomach and duodenum of rabbits treated with drugs like pilocarpine, which irritate the vagus. In one case a true peptic ulcer developed and perforated. Eppinger and Hess (*Zeitsch. f. klin. Med.*, 1909, lxviii), who have recently conducted extensive researches on the antagonism between the sympathetic and the vagus nerves in respect to their response to atropine and pilocarpine, found it possible to reproduce artificially with drugs the clinical symptoms of abnormal excitability of the vagus and also to inhibit with atropine both the natural and the induced symptoms. These investigators, who coined the term "vagotony" for this condition of abnormal excitability of the vagus, noticed the frequent coincidence of the condition with gastric ulcer and found that the pain with a gastric ulcer is peculiarly intense in "vagotonicers."

I have dwelt upon the influence which the central nervous system may exert in producing gastric irritability with pain because I believe that in many cases no treatment of hyperchlorhydria can be really curative that does not take this influence into consideration. Hyperchlorhydria is merely a symptom, a functional disturbance, the cause of which differs widely in different cases. The treatment should be begun, therefore, by endeavoring to remove the cause, although this is often more easily said than done. It is above all necessary to scrutinize the habits of the patient and to search thoroughly the various abdominal organs for centres of irritation. Aside from organic disease of the stomach itself, such as peptic ulcer, incipient chronic catarrh, and gastrectasis, hyperchlorhydria is frequently related etiologically to chronic disease of the gall-bladder or appendix, or to some affection producing constitutional disturbance such as chronic cardiac or renal disease, or chlorosis. Apparently constipation itself may be an important factor, although doubtless in the majority of cases it is the result rather than the cause of the hyperacidity.

In approximately one-half of the cases there is no evidence of structural defects either in the stomach itself or any of the adjacent organs, and the condition must be regarded as a perversion of function *per se*. Examples of this type are most frequently observed in young adults, both male and female, engaged in professional or clerical work. Mechanics and laborers furnish but a small proportion of cases. Indiscretions in eating and drinking, the excessive use of tobacco or alcohol, and above all, mental or emotional strain, are the principal causes. Obviously, therefore, these should receive the most careful attention.

In hyperchlorhydria from brain tire, travel, properly directed, often accomplishes far more than any method of medicinal or dietetic treatment, and may be confidently recommended to those who can afford it. In the majority of cases the chief aim must be to prevent any direct irritation of the stomach and to allay the morbid exitability of the nerve centres, or while attempting this to impair the conductivity of the nerves conveying impulses from the central nervous system to the stomach. Regular meals, regulation of the temperature of the food and drink, thorough mastication, avoidance of excesses in eating, drinking, and smoking, are cardinal points. Thorough mastication is especially important, for the more comminuted the food, the more bland it is, the more acid it binds, and the shorter its stay in the stomach. The alkalinity of the saliva is too slight to have much neutralizing effect, but the larger amount of mucus entering the stomach in consequence of thorough insalivation may be of advantage in this respect, as it is known that the mucus normally combines with one-fourth of the hydrochloric acid secreted.

Divergence of opinion still exists upon the question whether the diet should be composed chiefly of proteids or of carbohydrates. Penzoldt, Riegel, Meyer, and others advocate large quantities of proteids, while von Sehlern, Hemmeter, Bachmann, and others favor a preponderance of carbohydrates. Proteids, as has been shown, produce more acid, but at the same time combine with much of the excess and thus neutralize it. On the other hand, carbohydrates, while they call forth less acid, have no neutralizing power. Theory aside, the experience of actual practice seems to have demonstrated that although the patient is temporarily more comfortable upon a diet composed largely of proteins, no form of diet, proteid, carbohydrate, or mixed, has much power to reduce permanently the secretion of acid (Riegel, Kauffmann, Steele, Friedenwald, and Fischer). Therefore, the aim of treatment from the dietetic standpoint should be to improve the general nutrition, and thus lessen the morbid impressionability of the nerve centres, and at the same time to avoid everything that is likely to irritate the stomach directly. This is best accomplished by a liberal mixed diet of nutritious, bland, easily digestible food.

Vegetables poor in cellulose are the most suitable, and these are best taken in the form of purées. Tea and coffee should be used sparingly, if at all. Alcohol is generally inadmissible. Water-drinking at meals, provided it is not excessive and the temperature of the water is not too low, is not objectionable and may even act favorably by diluting the gastric juice. Boas (*Therap. Monats.*, May, 1906) regards it as an essential part of the treatment. Excessive smoking is distinctly harmful, but the question of cutting off tobacco entirely needs to be considered for each individual case. Foods to which vinegar or spices have been added and highly seasoned dishes of all kinds must be interdicted. Fat has a beneficial influence. When administered as a medicine in the large amounts sometimes recommended patients often turn against it, but when taken liberally as food in the form of butter, cream, and olive oil, it usually proves acceptable and efficacious. Fat not only depresses acid secretion. as Penzoldt, Boas, Pawlow, Bachmann, and others have showed, but it also causes an actual reduction of acidity by inviting a regurgitation of the alkaline duodenal contents into the stomach—a phenomenon made use of by Boldireff (*Centralbl. f. Physiol. u. Path. d. Stoffwechs.*, 1908, No. 6), Farr (*Jour. Amer. Med. Assoc.*, December 11, 1909), and others in obtaining duodenal secretions by way of the stomach. Boldireff believes that when fatty food is taken gastric digestion is carried on largely by the ferments of the pancreas, and further, that these ferments may be a factor in the production of some forms of gastric ulcer. Whether fat has any other function in the stomach is not definitely known, but it is not improbable that it also acts mechanically as a demulcent protecting the stomach from the action of irritant food and secretion.

Of drugs, the most suitable are those which protect the stomach directly against irritation, those which neutralize acidity, and those which diminish the abnormal excitability of the nerve centres or nerves. The best representatives of the first class are bismuth subnitrate and silver nitrate. Bismuth subnitrate is chemically inert and acts purely in a physical way by protecting the stomach against particles of food and irritating secretions. It may be given in doses of 20 grains or more, half an hour before meals. Silver nitrate probably also acts mechanically by forming in the stomach a thin coating of silver albuminate. It may be given in doses of $\frac{1}{4}$ of a grain half an hour before meals.

Of the alkalies, the most suitable are sodium bicarbonate, magnesia, and calcium carbonate. The ammonia preparations, owing to their stimulant properties, are undesirable. Sodium bicarbonate acts promptly and has no action on the bowel. It has the disadvantage, however, of giving rise to the formation of large quantities of $CO_2$, which may lead in time to distention of the stomach. Another objection that has been urged against sodium bicarbonate is that sodium chloride formed from it in the stomach tends to increase the secretion of hydrochloric acid, but this objection is probably not valid, as the amount of sodium chloride formed is very small and there is under ordinary conditions more of the salt present than is required in the production of acid. Magnesia has an advantage over sodium bicarbonate in that it neutralizes more acid. It is especially useful when hyperacidity and constipation coexist. Calcium carbonate may be substituted for sodium bicarbonate or combined with it when the hyperacidity is associated with a tendency to diarrhea. There is no experimental evidence to support the belief held by many writers that alkalies in themselves increase the secretion of acid. Reichmann (*Archiv f. Verdauungskrank.*, i) has shown that in man distilled water increases the free acid about as much as an equal amount of alkaline solution. Alkalies should be given at the height of digestion and, as a rule, in doses proportionate to the protein content of the meal; the smaller the amount of protein ingested, the smaller the amount of alkali needed.

Of sedatives, the most efficacious are the bromides and belladonna. The bromides have been very generally recommended. They are particularly useful when the hyperchlorhydric syndrome occurs as part of a wide-spread nervous erythism. In many cases strontium bromide is better borne than other bromides and is equally effective. Opinions differ as to the value of belladonna. When given indiscriminately it is, perhaps, as likely to fail as it is to succeed. Its virtue depends apparently more upon its power to lessen irritability of the vagus and prevent spasmodic contractions than upon its power to depress the secreting fibers of the vagus and diminish the secretion of gastric juice. Belladonna seems to have the peculiar property of arresting cramp-like contractions in the alimentary canal without interfering with normal peristalsis. Thus, it arrests the griping pain which active purgatives tend to induce without inhibiting in any way the movements which these drugs excite in causing evacuation of the bowel. It may be that the nervous mechanism which presides over tonicity and the violent contractions productive of pain is somewhat different from that which presides over normal peristalsis. This independence of tone and peristalsis has only been recognized since the introduction of the *x*-rays as a means of investigation. As Hertz (*Brit. Med. Jour.*, February 3, 1912) has shown, normal peristalsis is frequently observed in atonic and dilated stomachs, and in rare instances peristalsis is diminished in hypertonic stomachs. The cases of hyperchlorhydria in which belladonna or atropine is especially serviceable are those which display the symptoms of abnormal excitability of the vagus (vagotony), to which Eppinger and Hess have recently referred, or the phenomena described by Gowers (*Borderland of Epilepsy*) under the heading "Vagal and Vasovagal Attacks." The drug may be given in the form of the extract in doses of $\frac{1}{8}$ to $\frac{1}{4}$ grain, three times a day, or as atropine in doses of $\frac{1}{120}$ grain, more or less, two or three times a day.

# THE CESAREAN OPERATION; ITS WIDER APPLICATION; WITH REPORT OF CASES[1]

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After the introduction and establishment of antisepsis, and later asepsis into the practice of surgery, its application to obstetrics was devoted for the most part to efforts at sterilizing the birth canal. Not only when some operative measure was employed, but also in the natural process of labor, antiseptic douches were given. Time and experience led to the early abandonment of the latter practice, and the great gains to the obstetric art were for the most part confined to the application of the principle of cleanliness to whatever procedures had been in vogue up to this period.

The Cesarean operation done at first only on dead women to quickly rescue a living baby, and for the past three centuries on living women who presented some insuperable obstruction in the birth canal, has rarely been resorted to before the present century for other causes than obstruction due to deformities of the bony pelvis or tumors of the soft parts. Emboldened, however, by the experience of the gynecologist who showed that the peritoneal cavity and the uterus itself could be opened without great risk, the obstetrician began to resort more frequently to the abdominal operation for delivery. From March, 1835 when Professor Gibson of the University of Pennsylvania, did the first successful Cesarean operation ever done in Philadelphia, and about two and one-half years later repeated the performance successfully on the same patient, until 1880 no successful operation of this kind was done in Philadelphia. In this year Dr. Elliott Richardson did the Porro operation successfully. Dr. Gibson's operations were done before the days of ether, and the wound was not sewed up.

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[1] Read at a meeting of the Philadelphia Obstetrical Society, March 4, 1915. Reprinted from the American Journal of Obstetrics and Diseases of Women and Children, 1915, vol. lxxii, No. 2.

In 1882 Sänger announced his improved technic for the operation which included sewing of the wound and bringing the peritoneal surfaces together; and from that event the modern classical Cesarean section dates. It was about this time also that general surgical technic began its rapid strides due to the introduction of Listerism, and as laparotomy became less hazardous and hence more popular, obstetricians began to think more along surgical lines. In 1888 Dr. Howard A. Kelly did a classical Cesarean section on a woman with an external conjugate of 15 centimeters who had been in labor two weeks and whose bag of waters had broken four days before operation and saved both mother and child, the first to recover in Philadelphia since Dr. Gibson's notable case. From this time forward operations became more frequent, but they were done chiefly for some obstructive cause, and too often, as in this case, undertaken as a last resort thus leading to a high mortality. When it was realized that section of the pregnant woman was a very safe procedure, if done before she was infected by examinations or had spent herself in fruitless labor, the operation entered upon a new era, and the rights of the unborn child came to receive a consideration not heretofore accorded it. So from this time forward the Cesarean operation was rapidly adopted where formerly the child would have been destroyed, as in cases where tumors, ovarian or fibroid in the pelvic cavity, or cancer of the cervix, rectum or vagina or bony deformities obstructed the birth canal, and also where marked disproportion existed between the passage and passenger.

The mortality of this operation, which up to 1881 was 58⅓ per cent. according to Harris' statistics of 120 operations done in the United States, has grown steadily less, by reason of improved technic and earlier operation, until the present time, when it may be said to be on a par with abdominal section for any other cause. That is to say, in selected cases and in competent hands the mortality should be about zero, the operation *per se* being quite free from risk; but, of course. our patients do not always present themselves to us in this much-to-be-desired condition, but are brought into the hospital after being in labor many hours and even d2ys under the care of a midwife, and oftentimes after unsuccessful efforts at delivery by physicians. and are in consequence poor subjects for surgery. A 10.7 per cent. mortality in 571 abdominal Cesarean sections has recently been reported by Dr. Davis of the New York Lying-In Hospital where cases good and bad are received. It is not unreasonable to assume that this 10.7 per cent. mortality, *i. e.*, sixty-one deaths could have been reduced to 1 or 2 per cent.—if not entirely abolished —if these patients had been attended in the first place by a competent obstetrician who would have foreseen the troubles, and by timely interference ended them. Here is a rich field for an educational crusade, such as has been conducted with respect to appendicitis and tuberculosis. The laity should be taught that the risks attending parturition can be, for the most part, avoided by intelligent oversight of the parturient; by preparedness for rendering aid when needed, no less than by masterly inactivity when normal.

There is at present a growing tendency to broaden the indication for the Cesarean operation. Measured by the rule that that method of delivery which offers best chances for both mother and child shall be adopted, one will in these days more often than hitherto resort to section rather than delivery through the natural passages. I hasten to qualify this statement by saying that this presupposes a surgical training and proper environment for clean surgical work;

and is therefore largely a subjective question with the accoucheur. Included in this class are certain placenta previas, eclampsias and high forceps cases. Take for instance an eclamptic, at or near term pregnant for the first time with an undilated birth canal, an unobliterated cervix, and a living baby. Its delivery can be accomplished, in my opinion, more speedily and safely and with less violence to the mother's tissues, if not less shock by section, than by forceful dilatation of the natural passage. Experience only will establish this and this is in process of making.

And so with placenta previa, if the placenta be centrally implanted, and the child viable, or better near term, we will surely be able to save many more babies and perhaps more mothers by section than by any method yet devised through the natural passages.

As to high forceps operations, who of us has not felt, after this difficult procedure, especially in primiparæ, resulting in paralysis or perhaps death of the baby in delivery, and deep laceration of the mother's soft parts, that it would have been better to subject the mother to a slight risk by section—surely not greater than that due to violence done by high forceps, with considerable certainty of getting a live and uninjured baby, and a clean wound in the mother?

Following is the report of nine Cesarean sections done during the past year.

CASE I.—*Mrs. W., flat pelvis, section, resulting in live baby and recovery of the mother*—was admitted to the Kensington Hospital for Women January 20, 1914, and operated upon January 22, 1914. Her first labor which lasted twenty-eight hours was terminated by the use of forceps during which the symphysis ruptured with a loud report, according to her physician's statement, and separated about an inch. The baby weighed 8½ pounds and suffered from hemiplegia following its birth, from which it recovered in about two months. The mother was confined to bed six weeks, and on arising felt as if she was falling apart, and had great disability in walking, in spite of the pelvic support which she wore for six months or more. She suffered for a long time with pain in the symphysis and back, and for four months after labor had incontinence of urine. When, after two years and seven months she found herself again facing the problem of delivery, she naturally dreaded the ordeal very much, and begged for relief by some other method than the threatened forceps delivery; so she was admitted to the hospital two weeks in advance of term for the purpose of inducing labor, the head being movable above the brim. However, after discussing the situation with her physician (Dr. Galbraith) and husband and herself, the Cesarean operation was elected and performed January 22, 1914. The patient made a good recovery and went home in two weeks and suffered none of the disabilities and invalidism that followed the previous labor.

CASE II.—*Mrs. D., nephritis with marked edema and cough—section resulting in live baby and recovery of the mother*—was admitted to the Kensington Hospital for Women, March 11, 1914, with blood pressure of about 200 mm., extensive edema of the legs and face, skin of marble hue, mental dulness, headache and scanty urine containing albumin and numerous granular casts. Her condition improved considerably in a week by the use of a milk and buttermilk diet, purgation and baths. The urine, however, continued scanty, the twenty-four hours' output for the 15th, 16th, 17th and 18th being 12, 25, 15, and 26 ounces respectively, and after a distinct gain during the first few days after admission to the hospital, she began again to lose ground even under strict treatment; so on the 19th it was decided to bring about delivery even though no convulsions had occurred. Inasmuch as she was a primipara with breech presentation as determined by abdominal palpation, abdominal section was elected in preference to induction of labor through an undilated birth canal. Owing to her cough chloroform was employed as an anesthetic and a 10-pound baby quickly delivered. After the uterus was emptied it was observed that there was a marked projection of the sacral promontory, which would have greatly increased the difficulty of delivering a 10-pound baby, presenting by the breech through the natural passages. Her operative recovery was uneventful. Her general health, I am told by her physician, has been poor, owing to her nephritis.

CASE III.—*Mrs. S., Polish. Eclampsia—section resulting in a stillborn baby and recovery of the mother*, was admitted to the Episcopal Hospital, February 25, 1914, and discharged March 25, 1914. She was pregnant about nine months and during gestation had been habitually constipated, suffering during the early months with nausea and vomiting, and during the last four months with headache in the mornings and during the last two weeks with headache constantly. No treatment relieved the headache. She went into labor at 11 P.M., and at 1 A.M., had her first convulsion. Before she was admitted to the hospital she had had seven convulsions, one occurring in the ambulance. On admission she was edematous and examination showed her to have a small, nulliparous vagina, a relatively hard and undilated cervix. The baby's head was freely movable at the inlet and the bag of waters unruptured. Fetal heart sound were not heard. On account of the undilated birth canal, the unruptured membranes and the freely movable head, delivery by section instead of through the natural passages was decided upon. The patient was given ether, the vagina cleansed, the cervix painted with tincture of iodin, and iodoform gauze inserted into the vagina. The abdomen was prepared by the benzine and iodin method. A median incision large enough to turn out the uterus was made and a stillborn child was delivered. The patient made a good recovery.

CASE IV.—*Eclampsia resulting in a live baby and recovery of the mother.* Mrs. H., aged nineteen, primipara, admitted to the Episcopal Hospital February 7, 1914 pregnant eight and one-half months. The patient states that she was well during gestation, the kidneys were active and she was free from headache. The onset of the illness was sudden. She was brought into the hospital in the ambulance in convulsions with a pulse of 138, respirations 38 and temperature 100.2 °F. She was markedly edematous, especially the vulva. Examination showed a nulliparous vagina an undilated cervix and a live baby, wherefore delivery by section was decided upon in preference to forced delivery by the natural passages. The result was a live baby and a live mother, whose convalescence was partly febrile.

A considerable amount of ascitic fluid was found in both of these eclampic cases and none in the others.

CASE V.—*Nephritis, cardiac dilation, edema of the lungs. Section, followed by death of mother and baby.*

Mrs. G., aged twenty-four, primipara, admitted to the Episcopal Hospital March 10, 1914, and died March 12, 1914, pregnant eight months, has suffered a great deal throughout her gestation from headache and constipation. Ankles and eyelids are edematous. For the past three weeks has been very short of breath, with cough and expectoration of a mucoid material. This morning she grew suddenly worse. She could not lie down; she became deeply cyanotic, and was constantly coughing up a frothy and bloody mucus, which was her condition on admission. The respirations were rapid and labored, the vessels of the neck engorged; the pulse very rapid, irregular and weak; the right heart was dilated and moist râles were audible all over the chest. Twenty ounces of blood was withdrawn at once from the median basilic vein, and atropine, gr. $\frac{1}{150}$, strychnia, gr. $\frac{1}{30}$ and camphorated oil, ♏ xx., given hypodermatically followed by some relief. Prompt delivery of the baby was regarded as imperative, and owing to the primiparity of the mother, section was decided upon as the quickest method of delivery. Morphia gr. $\frac{1}{4}$ was given hypodermically, and the section done under local anesthesia for which Schleich's solution was employed. The abdominal incision only required an anesthetic. For the uterine incision no anesthetic was used or needed. A well-developed baby with pulsating cord was quickly delivered, but the assistants, in spite of faithful efforts could not succeed in establishing respiratory action. A large quantity of serum escaped from the baby's mouth and nose during the efforts to induce respiration, showing that it is probable that this infant sucked amniotic fluid into the bronchial tubes by intra-uterine respiratory efforts, stimulated by the blood highly charged with carbon dioxide which was furnished to it. Thus the baby appeared to be literally drowned in amniotic fluid. Following the operation which was done about midday the patient was quite comfortable with improved pulse, able to lie flat and relatively free from respiratory difficulty. Although pallid and waxy in appearance, her improved condition seemed to justify the hope that she might recover. She, however, passed only 2 ounces of urine during the first eighteen hours after operation, but 17 ounces were passed during the next eighteen hours; the abdomen became more and more bloated, the bowels resisting all measures for their evacuation. Toward evening nausea supervened, for which the resident physician passed the stomach tube, whereupon the patient had a convulsion and died about thirty-six hours after delivery. Her urine boiled solid with albumin and was loaded with granular casts.

Just how much effect the preliminary hypodermic injection of morphia had on the baby's respiratory function and in retarding the action of the mother's bowels can only be a matter of conjecture but it is the one feature in reviewing the case which I should like to omit.

CASE VI.—*Placenta previa. Section followed by recovery of the mother and death of the baby.* Mrs. D., aged thirty-six, married, six children and two miscarriages, admitted to the Episcopal Hospital March 20, 1914, pregnant nine months. Gestation was normal until the end of the eighth month when she began to bleed. The hemorrhages were small at frequent intervals, not attended with pain and without any known cause. On the night of admission, the patient had had an enormous hemorrhage, losing about 3 pints of blood. The attending physician packed the vagina without completely arresting the hemorrhage. On admission the examination showed the cervix partly dilated and occupied by the placenta. The fetal head was not engaged, but was freely movable above the pelvic inlet. The bag of waters was intact and the fetal heart sounds feeble if heard at all. On account, therefore, of the placenta previa, the floating head and possibly live baby, delivery was undertaken by abdominal section. Owing to the shocked condition of the patient, the pulse being 144, the presence in the lungs of râles and impaired resonance in the apices, nitrous oxide was given for the skin incision and for inserting the skin sutures. All other details of the operation were conducted without an anesthetic, the uterine manipulations causing no pain except where the organ was dragged upon. The baby's cord pulsated on delivery, but soon ceased; its respirations were never established. The patient made a mildly febrile convalescence, developing a superficial stitch abscess. Her abdominal preparation was made on the table by the benzine and iodin method.

CASE VII.—*Contracted pelvis. Cesarean section, recovery of the mother and death of the baby.* F. G., aged twenty-eight, admitted to the Episcopal Hospital February 27, 1914, has provided for the surgeons a veritable mine of abdominal pathology. Her first labor about one year after coming to this country was terminated by the use of forceps. The baby lived only about two weeks. In October, 1910, she had a gastro-enterostomy for gastric ulcer done by Dr. Charles H. Frazier. In June she had a Cesarean section done by Dr. E. P. Davis, resulting in a living baby. Following this section the patient had pain and a constant discharge from the vagina. In September, 1911, she was curetted and had no menstrual flow after that until February 6, 1912. She then conceived and throughout the period of gestation she had numerous fainting spells, when everything got black before her eyes and she would fall down; attacks of cardiac palpitation lasting several hours occurred. These symptoms increased after the fifth month. Labor pains began before admission to the hospital and on the morning of the day of admission became very severe. Examination on admission showed the cervix obliterated, the os dilated to the size of a silver dollar, the head not engaged but floating freely in the abdomen; the fetal heart sounds audible, rate 140. Cesarean section (her second) was done and the patient delivered of a living baby with a large hydrocephalic head, double harelip and cleft palate. It lived only a few minutes. It was noted that omental adhesions of the uterus were general. The uterine incision in this instance was sutured with chromic gut, three interrupted linen sutures and one continuous linen suture to close the peritoneum. This was an emergency operation, and it may be that the abdominal preparation was not satisfactory. At any rate the stitches became infected, and in the course of the next few months, one after another of them came out and in the course of time two hernias developed in the line of incision. Her health, however, was good except for abdominal pains and in due course she became pregnant for the fourth time, and pleaded for an abortion. I prevailed upon her, however, to let nature take its course, promising at the next delivery to remove the organs so that she could not conceive again. She suffered throughout her gestation with abdominal pain and two weeks before her estimated term was brought into the hospital in active labor and prepared for her third Cesarean section. At the operation considerable adhesions had to be dealt with although they were neither so dense nor so extensive as one would expect from the antecedent history. The uterus was delivered through a long central incision and an opening made in its posterior face to avoid the placental site. The baby proved to be poorly developed, small in size and not of healthy appearance. It was with difficulty made to breathe and lived less than an hour. The operation was completed by doing a supravaginal hysterectomy, leaving in the only ovary that was found and closing the abdominal wound after excising the hernial sacs. She made a good recovery and has since been enjoying better health than for some years before.

CASE VIII.—*Contracted pelvis. Section resulting in recovery of the mother and living baby.* A. K., Polish, aged thirty-two, married. No previous pregnancies. Labor pains started at 10 P.M., November 25, 1914 and continued until 2 P.M., when the membranes ruptured and from that time until admission to the Episcopal Hospital the following day, the pains were less frequent and severe. Internal examination about midday showed the cervix well dilated but the head not engaged. External examination showed the back to the right, the heart sounds below the umbilicus and the head movable above the brim. Delivery by section rather than by high forceps was undertaken at once; and in view of vaginal examination having been made both outside and in the hospital, the transperitoneal or extraperitoneal method was employed, and a 9½-pound baby delivered. Baby and mother did well—except the mother had wound infection late, either due to her scratching the wound—or to hasty preparation (by the iodin method) of the abdomen.

CASE IX.—*Flat and contracted pelvis. Section resulting in live baby and recovery of the mother.* A. B., aged twenty-eight. Married five years, pregnant the third time. At her first labor she was attended by a midwife, who after failing to deliver her called in a physician. By a difficult forceps operation he succeeded in delivering a baby which was so much injured that it lived only a short time. At her second confinement she was in labor twenty-four hours before admission to the Episcopal Hospital—unsuccessful efforts at delivery with forceps having been made by her physician. Here, I succeeded with great difficulty in extracting a dead and somewhat macerated fetus. So I advised her to never again have a baby at term delivered by the natural passages. She promptly conceived a third time and her confinement was dated for September 1. She came to the hospital a week in advance of this date and soon after admission the bag of waters broke before the onset of active labor pains, and with the head still freely movable above the pelvic brim. On the following day, I delivered her by the classical Cesarean operation and secured a healthy 7¼-pound baby. The mother's convalescence was uneventful.

To summarize briefly, these nine operations resulted in eight living mothers and five living babies—a record which if the operation were undertaken in the interest of the baby would render the procedure unjustifiable. This, however, was not the case. The mother's interest was paramount and the operation was thought to afford the quickest and safest method of delivery of the mother regardless of the baby's condition (in four of the cases).

When it was done deliberately for a contracted pelvis in a well patient, it was with a view to getting a living and healthy baby. One would not subject a healthy mother to this operation if her baby were known to be dead or dying.

It would be better to mutilate the baby and remove it through the natural passages.

Whether the morbidity and mortality following this radical method of dealing with these cases is justified, only the accumulated experience of a large number of operations and different operators will show—and toward that, this report is an humble contribution.

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